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A STUDY OF THE MUNITIONS INTOXICATIONS IN FRANCE.

By Roger G. Perkins, M. D., Professor of Hygiene and Bacteriology, Western Reserve University; Consultant in Hygiene, United States Public Health Service; and Medical Associate to the Scientific Attaché to the American Embassy, Paris, France.

[EDITORIAL NOTE.—This study was made under the auspices of the National Research Council and represents a great deal of time and effort on the part of the investigators.]

The discovery that many of the various explosives manufactured in Europe and the United States had more or less toxic effects on the workmen who handled them, with many fatal results, has led to a series of studies in the various countries, and to the appointment of various commissions and committees on munitions intoxications. The present attempt at analysis of the question in France was made at the request of the American committee and was carried on while the writer was Medical Associate to the Scientific Attaché at Paris, under the American National Council of Research. The unexpected arrival of the end of the war, with the consequent immediate cessation of activities, made the work, unfortunately, incomplete; but through the courtesy of the Ministry of Munitions it was possible to meet some of the most important men who had had the control of the medical side, and to see the places where the work had been done. It is fortunate that as far as di-nitro-phenol and tri-nitro-toluol are concerned the French consider the problem solved, so that there is a greater degree of completeness than had been hoped for.

The investigation has been particularly interesting on the one hand because di-nitro-phenol is particularly a French explosive, and on the other hand because the troubles which have been recorded in England and in the United States in connection with tri-nitro-toluol have been scarcely seen in France. For this reason the chief discussion in this paper relates to the di-nitro-phenol problems and the methods attempted for their solution, with briefer notes on the French opinions in connection with the toluol derivatives.

Statistics.—While there has been a great variety of explosives manufactured and tried out in France, the main ones have been di-nitro-phenol, tri-nitro-phenol or picric acid, and tri-nitro-toluol. Most of the filling mixtures have been various combinations of these, and the DD or 40-60 mixture of DNP and TNP is the most used. It is not possible at present to get the statistics on the amounts of

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the various explosives manufactured or the number of workmen employed except as will be found in the body of the paper, where is given the only accessible information relating to the number of fatal cases in relation to the 10,000-ton production of DNP in 1916–17 and in 1917–18. For the same reasons it is impossible to determine the number of whites, blacks, and yellows, though all were employed, or even to determine the proportions, and in the confusion resulting from the war condition and the changes of groups of men from one place to another, especially true of the Senegalese and the Anamites, it is improbable that such statistics will ever be available for study.

In the same way all the information which has been obtained must be taken with a reservation, as there are various complications which do not appear on the records. It was necessary to move the factory physicians from place to place to care for certain emergencies, so that there is often a lack of continuity in the information, and there was, of course, a great variation in the individual efficiency of the different men.

Another point which must be taken into consideration is that there were from time to time, and even from day to day, changes in the demands on the factories, the relative amounts of one or another explosive demanded altering in agreement with the demands from the artillery divisions. In some of the factories these changes necessitated the sudden transfer of men from one melt to another, the change of explosive being often made without change of shop after the cleaning of the vats. Under these conditions the urines of the men who had been on the DNP and were changed to another melt which did not involve DNP were no longer examined, so that the records of examination show apparent wide discrepancies, suggesting incorrectly that the records were improperly kept up. In connection with the Senegalese it must also be remembered that these men were differentiated by numbers and it was found that they had the habit of exchanging numbers or taking the number of a dead man or one who had left, so the records as regards them are practically worthless as statistics, though I am inclined to take the personal opinions of the close observers as essentially correct.

With these rather unsatisfactory preliminaries the question of the toxicity of the various explosives can be taken up, and the most important, namely, the di-nitro-phenol, will be first discussed.

I. DI-NITRO-PHENOL INTOXICATION.

While di-nitro-phenol is theoretically a double nitration of phenol, the more usual method of manufacture is from benzene, by the formation of mono-chlor-benzene, which by the treatment with nitric and sulphuric acids is changed to di-nitro-chlor-benzene. Further

treatment by NaOH substitutes NaO for the Cl(NaCl), and the Na is removed by treatment with HCl, leaving the completed di-nitro-phenol.

It is obvious that there are six isomeres possible, all of which are known, but the ordinary DNP is the isomere 1-2-4. A further nitration results in the formation of tri-nitro-phenol, which is again possible in six isomeres, of which the most common is the isomere 1-2-4-6 (the ordinary picric acid or melinite).

In the process of the nitration of the chlor-benzene there are formed also certain mono-nitro-chlor-benzenes (notably the para-, with some ortho-) which are not acted on by the NaOH. These must be washed out by a current of steam before the addition of the mineral acids. The finished DNP is a yellow or yellowish-white solid, brownish when improperly made, and with a faint odor of phenol. It can form fine crystals. When pure, the melting point is 114–115 C. It may be sublimed and carried off in aqueous vapor to the amount of about 2-3 grams of DNP to 2 kilograms of steam at 100 C. It is soluble in 21 parts of boiling water, and in about 250 parts of water at 18 C. It is also soluble in alcohol, ether, and chloroform. The reaction is distinctly acid, and crystalline salts are readily obtainable, as for instance, the sodium salt, $C_6H_3(NO_2)_2$ (ONa) H₂O.

If there is insufficient treatment with steam there may be a residue of mono-nitro-chlor-benzene, marked by a smell of anise which may be very strong. In the commercial product there may be also traces of chlor-5-nitro-2-phenol. In the nitration of the chlor-benzene there is a small amount of the 3-4 formed, and the NaOH does not attack its atom of Cl, but replaces the NO₂ in the position 3 by a hydroxyl group.

$$C_6H_3$$
 $No_2(3) + 2NaOH = C_6H_3$ $ONa(3) + NaNO_2 H_2O$
 $NO_2(4)$

It is considered, however, by the French that the various impurities are all less toxic than the DNP itself and can therefore be disregarded as essential factors in the clinical cases. (Appendix 7.)

In the process of manufacture the details are well known, but the essentials, as far as the present investigation is concerned, deal with those parts which admit of the access of the DNP in any form to the various portals of entry of the worker. From the chemical characteristics it may be readily seen that wherever sufficient heat is

used to cause volatilization, or the carrying off of appreciable quantities of the chemicals in water vapor which comes into contact with the workmen, we have one group of possibilities. The other group is in the handling processes, where the solid material comes into contact with the employee in such a way that it may remain on the skin long énough for absorption, or reach more indirectly any of the other portals of entry.

Portals of Entry.

According to the work of Guerbet and others, there are three portals of entry, the skin, the digestive tract, and the respiratory tract. The relative importance of these depends on the character of the work, as in different processes one may be more accessible than another. In general, it is the belief that the skin is the most important, though there are certain series of cases which, if correctly reported, appear to indicate that the respiratory tract is more important. There appears to be no question, however, that all these portals may be used by the intoxicating agent and that through them it may reach the circulation and set up the various specific disorders.

The work of Guerbet and Mayer shows that, in the body, the DNP undergoes certain chemical changes after reaching the blood, or even before, into reduction derivatives, which are of varying complexity. Examination of the blood, the organs, and the urines of fatally intoxicated men shows that while in some the blood and the organs contain unaltered DNP and the urine contains reduction products, in others all contain both the DNP and the reduction products. The main substances found in the urine are as follows:

DNP 1-2-4, eliminated as such;

Amino-2-nitro-4-phenol;

Amino-4-nitro-2-phenol:

Di-amino-phenol.

Nitrogen compounds resulting from the combination of two molecules of amino-nitro-phenol or of di-amino-phenol.

All these except the amino-2-nitro-4-phenol may apparently exist in the urine without any clinical signs of intoxication. This compound, however, while not certainly the proof of intoxication, has always been found in great abundance in the urine of the serious cases. It is accordingly this substance which is used as the basis of the specific test on the urine, known as the Derrien test, or the violet reaction of Derrien, noted elsewhere. (Appendix 1.)

Factors in Susceptibility.

With this diversity of portals of entry, the fact that only a limited number of persons has been affected even under the most unfavorable

working conditions indicates that there must be a very marked variation in susceptibility. Other things being equal, we have the questions of age, sex, and race as the primary possible factors, after which come the more individual characteristics dependent on habits and on physical condition.

Age.—This can practically be neglected, as all the workmen were within the mobilization age and there are no indications that those who were at the later limits of that age were more susceptible than those at the earlier periods, or vice versa. There was no child labor at all. It must be remembered that this refers to the sections of the factory where the actual manufacture and handling of the explosives took place, as there were other sections of the factories where there was no exposure to intoxication.

Sex.—Inasmuch as there was a ruling that no women were to be employed in the dangerous processes, there have been very few employed, and practically all these were in work which was not dangerous. There are no records of any cases of illness among women employed in the jobs exposed to the fumes, vapors, or contact.

 \bar{R} ace.—There are certain interesting points in connection with the race factor in susceptibility, as the employees not only came from various parts of France, but also included Annamites and Chinese representing the vellow races, and Senegalese representing the black In those factories where the records were carefully kept there was accordingly an opportunity to compare the relative susceptibilities. In general it was found that the Annamites were the least affected, the Senegalese next, and the whites most; but this is to be taken with caution, as other factors of importance may have a very definite bearing. For instance, the Anamites are acknowledged to be the most careful in following the regulations, especially as regards the fumes, and the whites to be the most careless. The question of the relative alcoholism is also very important. It must, however, be noted that among the Senegalese there was frequently a very marked Derrien test of a more or less transitory character, without any serious clinical indications.

At Sorgues, near Avignon, Dr. Senglars states that there were very few cases reported among the Senegalese, although it was not infrequent to find a very marked Derrien test, giving the impression that the blacks are relatively insusceptible. This is, of course, in agreement with the recent experiments in America showing that the colored race has a greater skin resistance to mustard gas than the whites (personal conversation with H. G. Wells), but in the case of the Senegalese the results must be taken with caution. We find that while the whites were under control of the French doctors, the Senegalese were cared for by Colonial doctors, many of whom had comparatively little aptitude for the study of these complex problems,

and cases may easily have been missed. This is the more likely, as the Senegalese were very susceptible to pneumonia and many died of this disease with each change of weather. In the press of work the diagnosis was probably more or less inaccurate, and the general diagnosis of pneumonia was made on inadequate grounds. At least one case was seen on which no diagnosis of DNP intoxication had been made, but which when seen had the characteristic abundant sweats, and when this case was treated by bleeding and other specific methods, it recovered. One of the attendants stated that there had been several similar cases which had been diagnosed and treated as pneumonia and had died under that diagnosis. Other trustworthy observers who have actually made physical examinations of the supposed pneumonia cases report that auscultation was negative and that diagnosis apparently rested entirely on the elevation of temperature and the prostration. It seems probable that the resistance in the various races was practically identical.

Alcoholism.—The one point on which there is no disagreement is that the men with an alcoholic history either past or present were by far the most susceptible and had to be weeded out at once. The districts in France which have the reputation of housing the heaviest drinkers also supplied the heaviest toll of DNP intoxications. This statement is not checked by accurate information but it is the opinion of each of the men in charge of the works visited in different parts of the country.

Physical condition.—(a) Lesions of the liver and kidneys.—This factor is, of course, closely allied to the question of alcoholism, but also includes other cases which have a different etiology. In general, all those persons with the presence of albumin in the urine show a marked susceptibility and are to be included among the serious risks. The actual statistics with regard to the urine are available but it has been the regular practice to remove from the job all men having any urinary troubles apparent in the preliminary urine examinations. There is less evidence in connection with the liver lesions, most of the facts being obtained at post-mortem, and no accurate statistical results can be given.

- (b) Respiratory tract lesions.—There has been no relation of the intoxications traced to these even when the apparent portal of entry has been the lungs, save that tuberculosis is a predisposing factor. This, however, is more probably related to general physical condition than to local lesions.
- (c) Digestive tract lesions.—There is no evidence that preexisting lesions have any bearing on the susceptibility.
- (d) Cutaneous tract.—There is no evidence that lesions of the skin have made the absorption of DNP easier, and, indeed, it appears

probable that such absorption takes place through the normal structures and would be checked rather than aided by lesions.

(e) General physical condition.—It appears that the resistance in persons with a low grade of physical condition is less than in the healthy, and this is emphasized by the fact that in many cases where the workers have been apparently resistant over long periods of time, if they are sick or overworked they may suddenly develop symptoms. It was partly on this basis that the administration of milk to the workers was started.

Clinical History.

The clinical history has been well worked up by Prof. Etienne Martin, who has had the opportunity to see the cases all over the country, and a summary follows.

1. SUBACUTE INTOXICATION.

This is especially important in calling the attention of the attending physician to the dangers of a more severe attack, enabling prophylactic removal of the worker.

- (a) Gastro-intestinal troubles.—These are the most frequent, and include anorexia, with a white and furred tongue, followed by nausea and vomiting; there may be diarrhea and colics. It is only exceptionally that there is icterus.
- (b) General symptoms.—Workers claim that they have grown thin to a notable extent after several months' work in DNP. Many complain of general weakness with headache and dizziness, with moderate sweats, especially at night. A few days' rest are usually sufficient for a complete cure. The urine shows a positive Derrien, and when this increases day by day or remains at a fairly high point it is an indication that an intoxication of the acute type is about to develop.

2. ACUTE INTOXICATION.

This is generally a sequel to the subacute symptoms, and especially to the gastro-intestinal signs. The onset is sudden, with complaints of having the arms and legs "cut off" (very tired). There is a painful constriction at the base of the chest, and a burning thirst.

The face is pale with slight cyanosis of the lips; there is abundant sweat, and a characteristic agitation and axiety. The respiration is short and dyspnœic, and according to Dr. Senglars at Sorgues, the difficulty is in the *inspiration* in contrast to the *expiratory* difficulty in asthma. There is a moderate elevation of temperature, with a regular pulse. With the occasional exception of a few râles at the bases, the lungs are found to be clear.

There is a marked diminution of the quantity of urine, and a positive Derrien of increasing intensity. Improvement in the case is marked by a marked increase of urine (spoken of by many as a "débacle urinaire").

Removal from work, with a rest cure free from exposure to the intoxicating material is usually followed by a rapid cure. It is to be noted that a single attack of this sort is in no way an immunization, but that these men require careful watching.

3. FILMINATING INTOXICATION.

This is especially noted among alcoholics or persons with renal or hepatic troubles. Death may supervene in a few hours. The usual course of the disease is as follows:

Sudden onset of advnamia, with inability to continue work, or, less frequently, violent colics and abundant diarrhea. After leaving work and going home the condition is aggravated, there is an elevation of temperature up to or even exceeding 40° C., there are abundant sweats, which stain the skin yellow even in the places where there has been no exposure of the skin to the chemicals. There is intense thirst. At times there is an apparent improvement after a bowel or bladder discharge, giving false hope of recovery, while the heart remains regular and auscultation shows nothing except occasional scattered rales. The pupils are contracted, the patient is frightened and excited, and partial or general convulsions follow. This condition of excitement is followed by unconsciousness, coma, and death in a few hours. It is a clinical picture of the end of a fatal uremia case. One of the conspicuous points after death is that the extreme dehydration of the tissues leads to very early rigor mortis, with delay of decomposition of the cadaver.

Some of the cases are even more definite, the workman complaining of the various subacute symptoms, but staying through the work time. He is found somewhere along the road breathing with difficulty, covered with sweat, with a temperature of 41° C. or even 43° C., and dies before anything can be done. In these fatal cases there is a rise of temperature after death, sometimes of several degrees. Urine obtained by catheter shows an intense Derrien.

POST-MORTEM EXAMINATION.

Perhaps the most interesting feature of the post-morten examination is that there are no lesions to be found which are in any way characteristic. The only thing is the acute ædema of the lungs determined by the intoxication of the vasomotor system, obviously the cause of the respiratory difficulty. The microscopic lesions in the liver and kidney cells are inconstant, nor are there anywhere else

any typical changes. It is true that there are readily found in the blood and the organs traces or even more of the DNP and of its derivatives, but this is also true in the nonfatal cases. Moreover, the workmen who die from accidental causes while employed on DNP show the same thing, so no great weight can be placed on it. The most that one can say is that when workers in DNP develop these characteristic symptoms and die after the usual period or are seriously ill and recover under proper treatment the illness is due to the DNP. One may also say that where there are lesions of the renal or hepatic system the resistance is lessened, and this is also true when there is associated tuberculosis, malaria, or chronic rheumatism. The lack of resistance of alcoholics has already been noted and requires only additional emphasis here.

Experimental Work.

Under the Conférence pour l'Etude de la Toxicité des Explosifs (Report in MS.), established in 1915 after the appearance of serious results from the manufacture of DNP, a long and elaborate series of animal experiments were begun by Dr. André Mayer, assistant director of L'Ecole des Hautes Etudes, and completed by him in 1918. The investigation covered experimentation with all sorts of animals, both warm and cold blooded, and also with man, including attempts at therapeusis of various sorts. The author's summary gives an excellent résumé, a translation of which follows:

INTOXICATION IN ANIMALS.

Toxicity of di-nitro-phenol.—The long experience of the Service of Explosives in the manipulation of picric acid, of which hundreds of tons have been made and used without serious cases of intoxication, led to the idea that the nitrated phenols are not violent poisons for the human organism. Accordingly, when the manufacture of dinitro-phenol was begun and the first cases occurred among the workmen they were put down to impurities in the commercial products. This idea was shown to be incorrect. All the impurities which it was possible to extract and even all the various compounds of benzene formed in the course of the manufacture were found to be less dangerous than the di-nitro-phenol 1-2-4. (Appendices 6 and 7.)

This, then, is a toxic product, no matter how introduced into the animal organization, whether by ingestion, intravenously, subcutaneously, intraperitoneally, or even when rubbed on the skin. This is true for all the animals tested, namely, the horse, dog, rabbit, pigeon, turtle, and frog. In all of these the toxic dose is 0.01 gram per kilo of animal.

Acute experimental intoxication.—Di-nitro-phenol is not only a poison, but a specific poison. The symptoms are characteristic and have a common basis among all the warm-blooded animals. In the first place, there is a considerable exaggeration of the heat-radiation activities as shown by a thermic polypnea in the dog, vasodilatation

and sweats in the horse. In the second place, in spite of these reactions there is a progressive and considerable elevation of the temperature, which may rise to 45° C. at the time of death. In the third

place, there is an immediate rigor mortis.

The fundamental phenomenon is an extensive increase of the combustions, which is neither directly nor indirectly the result of a stimulation of the nervous system. It occurs even in the cold-blooded animals. It bears no relation to an increase of muscular work; it is general and does not appear to result from any indirect action on any special organ, but from a direct action on the general economy. In fatal doses then, the poison appears to be a general stimulant of the cellular oxidations.

The fatal intoxications naturally affect the general nutrition. During the intoxication one notes modifications of the metabolism of sugar (disappearance of the reserves of glycogen, hyperglycemia). One may also suspect variations in the metabolism of the fats (variation of the chemical composition of the various organs), but only to a limited extent. No changes can be seen in the excretion

of nitrogenous or saline derivatives.

Furthermore, the fatal intoxications produce functional alterations in certain organs, especially in the liver, whose cytologic structure is transformed (variations of the chondriosome, appearance of abnormal inclusions), together with a change in the chemical com-

position.

Nonfatal experimental intoxications, if the dose is fairly large, may cause similar symptoms, while with feeble doses these may be entirely absent. But even in these cases the intoxication has an effect on the general nutrition, modifying the nitrogenous and sulphur eliminations. It may also alter the functional value of im-

portant organs like the kidney.

Chronic experimental intoxication.—In the same species of animal there is a variable susceptibility to the poison. A few animals succumb to a dose below the normal, and a smaller number resist a dose one and a half times the normal. When a series of nonfatal doses is given, a tolerance to the fatal dose is acquired by the majority of animals. The tolerance is established rapidly and the animals can be daily given a fatal dose for a fresh animal for periods of as long as a month and a half. This indicates that there is no accumulation and that a definite concentration in a single dose is necessary.

There may be no characteristic symptoms in this chronic poisoning, but there are always increases in the exchanges. Sometimes there are polyurias, phosphaturias, and even lesions of the liver and the

kidnev.

Alteration of the di-nitro-phenol in the body.—In the blood and in the organs are found either the unchanged substance or its derivatives, the amino-nitro-phenols (amino 2 and amino 4) and sometimes the mono-nitro-phenols. In the case of acute intoxication the amino 2 nitro 4 phenol is always present.

INTOXICATION IN MAN.

The symptoms of intoxication in man seem closely bound up with those in the animal experiments. In the cases which are to develop fatally there are premonitory malaises, digestive troubles, profuse

sweats, dyspnoea, agitation, elevation of temperature, and after death there is early rigor mortis. The intoxication may begin suddenly, and death may occur a few hours later. The symptoms in the severe cases which get well are much the same at first, but the second or third day shows marked improvement with rapid recovery.

At autopsy there are no characteristic lesions. There may be

cedema of the lungs; at times a fatty infiltration of the liver. The blood and the organs always contain the DNP or its derivatives and the urine always contains large amounts of amino 2 nitro 4 phenol.

Therapeutics.—All attempts to relieve the conditions by increasing the heat output or by giving antithermics or reducing drugs have no effect. All that can be done is to try to give the body the means of passing the crisis. The best results have been obtained by the use of

massive intravenous injections of glucose or inverted sugar.

Pharmacodynamic specificity of DNP 1-2-4.—The administration of ortho or meta mono-nitro-phenol or of picric acid (tri-nitro-phenol) gives none of the above results. The 1-3-4- isomere of the di-nitro-phenol and the para-mono-nitro-phenol do cause them, but only with heavy doses, and even then in a transitory manner. The other isomeres have a very different effect. The 1-2-3- and the 1-3-5 lead to the change of the hemoglobin to methemoglobin. This indicates a very high specificity of the di-nitro-phenol 1-2-4.

As a result of these observations of Mayer, attempts at prevention can be largely removed from an empirical basis.

Mechanical prophylaxis.—Inasmuch as it has been clearly shown that all the usual portals of entry may be available for the entrance of the intoxicant, all possible measures must be taken for preventing the access of the DNP or its derivatives to the workman. In general, the material is in a dangerous condition when in the form of vapor or of dust or when the method of handling brings the solid in contact with the skin. It is also apparent that when the weather is warm the danger is increased. This is probably due to a greater permeability of the skin when there is a tendency to sweat, and to a higher vapor tension.

Whenever possible the processes are to be carried on in a closed apparatus, with the connections closed, so that there will be no release of vapors, and in many cases the improvement of the apparatus has enabled this to be done satisfactorily. On other processes it is still apparently necessary to bring the materials to temperatures where there are vapors and where the workmen must dip out the material or handle it while hot. Even with the use of overhead ventilators, with or without forced draft, there is a good deal of exposure to the vapors, and in such cases there are asbestos curtains around the vats, supposed to be raised only at the time of inspection or handling. In some places, especially where the material is dried in driers or where it is sifted or granulated, there is a good deal of fine dust, which permeates the atmosphere. Here the use of masks is indicated. The personal equation of the workman here, however,

is much as it is in the United States, and the masks are not worn, the curtains are not dropped, and the workman prefers to take his chance. It may be noted in this connection that the Anamites are by far the most careful and the most amenable to the rules, and incidentally it may be recalled that they are the least affected by the poison.

The use of gloves has been suggested, but it has been found that the use of rubber gloves is dangerous, as when the material gets in the glove the skin is macerated and the effect is additionally bad. When gloves are used it is probably better to use washable ones and to see that they are washed at frequent intervals. The use of salves has also been suggested and tried, but none has been found which is sufficiently permanent to be of use.

The insistence on the cleanliness of the operative is perhaps the one most effective mechanical means, but offers great difficulty. From the ideal standpoint the following routine should be employed:

Each worker should have two sets of clothes (both outer and under), one of which should be worn in the factory only, should be the property of the factory, and should be cared for and washed by the factory. When the worker enters he is to leave his clothes in one room, pass into another, and put on the working clothes; and whenever he leaves the factory he must leave the working clothes, pass through a bathroom, in which he takes an actual bath, and then put on the clothes which belong to him. In addition, a place other than the workroom must be provided in which he can eat, and the eating of food in any place where the toxic material is handled must be strictly forbidden and the rule enforced. Where possible there should be an enforced rule insisting that the hands be washed and the nails cleaned before eating. The great difficulty is that the workmen object to the use of water either externally or internally, and unless the arrangement is good and a competent man is in charge the baths will be passed by, the hands will not be washed, and food will be eaten on the sly. Another difficulty has been found in France in that the use of printed rules posted in the conspicuous places has immediately resulted in a great amount of neurasthenia, so that after such posting there has been a very large influx of patients to the doctor and a plentiful refusal to work in the explosive as soon as it is known there is danger. When this phase passes the rules are as though they did not exist. In some places it has been found possible to enforce the baths by the arrangement that in order to get from their working clothes to their street clothes it is necessary for the operatives to pass actually through water nearly up to the waist with the shower going above, and by the arrangement, in other places, that when there is no bath there is a reduction of 20 cents in the daily wage and when it is taken there

is a reward of 20 cents, making a prize of 40 cents for the clean and virtuous.

Broadly speaking, the efforts at cleanliness are at best only partial and act as adjuvants to the mechanical devices for the prevention of contact. The best means of prevention are probably those under the following heading:

Medical prophylaxis.—As noted earlier in this paper, it has been found that the reaction of Derrien is a fair indicator of the susceptibility of the worker, and in France it has been especially on this basis that the medical work has been done. For the proper performance of the test and for the carrying out of the subsequent treatment it is necessary to have a man skilled in urine examinations and with the proper laboratory equipment, one who is to work in association with a factory physician, to whom all the urinary records go for the determination of the further activities in each case. The factory physician should have facilities for complete physical examination and should, of course, be competent in diagnosis to differentiate from other ailments and to classify the symptoms noted above as suggestive of DNP poisoning. There should also be readily accessible a hospital or infirmary, where any serious cases could be treated at once. If these matters are cared for, the system is as follows:

At entrance there is a general medical examination as far as possible, with information as to history of alcoholism and of syphilis or kidney troubles, and a complete urine examination to determine the presence of any weakness of the kidneys. As far as possible, all men having any of the earlier noted predispositions should be refused admission. This is often impracticable, but, even so, the history will indicate which men need watching. Men with a history of malaria and with enlarged spleens and livers are also bad subjects. especially so if alcoholic. After the preliminary selection of the men there should be a weekly round by the doctor, during which he can note the conditions and see if any of the men are suffering from ailments which they are not reporting. This will also afford an opportunity to educate the men in the essentials and to show the reasons for the necessary care. At these visits he will also examine the urinary records for the positive Derriens. It is the opinion of Martin that where the dusts and the vapors are removed, where the workers are properly selected, and where cleanliness is enforced, there should be no positive Derriens. This, in other words, says that the presence of a Derrien in men working in a theoretically correctly run shop means that there is a fault in technic, either in the mechan-

ical devices or the hygiene of the workman, or that there has been a change in the personal equation of the individual. If a positive test is found and persists for several days, or increases in intensity, the workman must be examined at once, kept under surveillance, and removed from the shop as soon as there are any of the early symptoms, especially if there is any elevation of the temperature. "The progressive daily increase in the intensity of a Derrien is to be considered as a sign of intolerance." Inasmuch as there is no rule as to the exact relation between the intensity of the test and the danger to the individual, the intensity should not be allowed to pass the fourth or fifth degree (Appendix 1). The main value is the information to the attending doctor.

Inasmuch as for the establishment of a disintoxication there is a minimum of eight days, it has been suggested that there should be, as far as possible, a rotation of service so that the men will not work in the dangerous divisions for more than two weeks at a time, with a subsequent change for two weeks to a shop free from danger. This will also prevent the development of the cases in which after a period of freedom there is a reaction following overwork of an intensive character. There have been technical difficulties in this connection, and, as a matter of fact, it has been left largely to the individual factories to settle.

It should also be the duty of the visiting physician to see that the various rules are enforced, that ventilation and the withdrawal of the vapors and dusts are being efficiently carried on, that the clothing which is supplied to the workmen is of a suitable character, as, for instance, in the wooden sabots, which are the habitual footgear in France, there must be ankle pieces preventing the dust from falling in and being macerated into the skin of the feet. This, of course, has been noted in connection with the TNT workers in England. Against the powders and dusts of the DNP the Tissot mask of the special make for the munitions has been recommended.

Treatment.—In all simple cases the withdrawal of the workman from the danger area is sufficient. He should be kept out at least until the test in the urine becomes negative. In the more serious cases the treatment is still of the symptomatic character, as there is as yet no special antidote available. The most successful treatment consists in the attempt to get rid of the poison as fast as possible by the use of purgative, and by abundant bleeding followed by injections of glucose solutions. Martin believes that the bleeding is of value not only as a diluent of the poison, but also from its action as a vaso-constrictant, in neutralizing the vaso dilatation effect of the DNP. In connection with this treatment the administration by the

mouth of alkaline drinks and milk is indicated. The use of the latter is, apparently, mainly because of its value as a rapid nutritional factor, as I have found no one who offered any reason why it might be of value otherwise. The use of morphine as a check on the excitement and dyspnæa is also indicated. This treatment should be given in the hospital and the patient should be watched carefully till safe.

Martin considers the milk treatment as of no value, except as a food, and classes the demand for it and the violent objections to its disuse in the same class with the corresponding use and disuse of coffee in the works where there was nitroglycerine, and the coffee was used empirically and then abandoned. The protests were profuse, but the withdrawal had no ill effects; in fact the contrary, as the habit which had developed of taking so much coffee had produced bad physiological effects, and had actually reduced the efficiency.

Systematic Activities of the French Government.

The question of munitions intoxications was taken up very seriously by the Ministry of Munitions, and it is owing to their appreciation of the efforts of their experts appointed in 1915 that much of the improvement in the manufacturing conditions and the resultant improvement in the health of the employees has been possible. In all my inquiries they have shown the greatest courtesy and consideration, and I desire particularly to express my appreciation of the help of the following:

Inspector General of Munitions Vieilles.

Dr. Etienne Martin, Professor of Legal Medicine at Lyon.

Dr. André Mayer, Assistant Director in l'Ecole des Hautes Etudes.

Dr. Guerbet, Secretary to the Munitions Intoxications Commission.

Mr. Lheure, Director of the Manufacture of Explosives.

Dr. Senglars, Physician in charge of factory at Sorgues.

A brief account of the history and development of the commission follows, the original report of which is in MS.

Soon after the appearance of the cases of intoxication in 1915, there was formed, under the auspices of the Ministry of Munitions, a Commission for the Study of the Toxicity of Explosives. There was a large personnel established with very competent specialists and investigators, and a report was presented in June, 1918, covering the work of the commission. The following is a brief account of the plans and accomplishments:

As soon as a substance is noted as dangerous or likely to be dangerous, a systematic study is made and distributed to the members. At the same time investigations are carried on in the factories as a routine measure. Abstracts are made of the foreign work

which are studied by the conference. (Then follows a brief statement of the results obtained by Guerbet and Mayer and Martin, noted elsewhere in this paper.) Instructions were given in a special course at the Collège de France to the men who were appointed as specialists, and these men were then distributed to the various factories. The visiting physicians were ordered to make clinical examinations of the workmen, with regular routine visits, and specially trained pharmacists were appointed for the urine examinations. It was made their duty to examine frequently the urines of the workmen, every two days in hot weather, less frequently in cold weather, and to report the results to the physician in charge.

In addition, measures for the general hygiene of the workers were established, including the rotation of service and obligatory shower baths. The desiccators were given up and the product was manipulated in a moist condition to avoid dust, the fusion vats were covered with an asbestos curtain and ventilators were established above them, and the granulation which followed the fusion process and which was formerly a hand process, was made a mechanical one. There are in process of substitution for the granulation, which is dusty at the best, other procedures such as "drageification," resulting in somewhat larger masses and performed by the "tonnes Landrin," or poured in slabs, by the "cylindres Tesmart." Similar regulations and changes have been arranged for the loading factories.

Modifications of the law of 1898 on industrial accidents were made, which have the effect of adding the munitions intoxications to the other formerly listed accidents. Other studies were undertaken on the intoxications by nitrous gases and vapors, and the use of the army gas masks, with modifications in the metal parts, was recommended. A number of these were accordingly made, and the workmen are provided with them.

The results of these activities have been satisfactory, and while the records are not of a sort which gives accurate statistics of the morbidity and mortality in comparison with the numbers employed, by means of comparing the number of deaths per 10,000 manufactured tons in the year prior to the establishment of these improvements with the number in the year following, some idea can be obtained.

Period.	Deaths.	Tons of DNP and mixture DD manu- factured.	Deaths per 10,000 tons.	
May, 1916, to May, 1917	31	19,100	16.3	
	5	40,700	1.2	

Through the courtesy of Dr. Guerbet, the actual records of the various factories in connection with the use and results of the Derrien tests were placed at my disposal and served to confirm the statements of the various men to whom I am indebted for my information. This is the more valuable, as the records had not yet been analyzed in any detail and no graphs had been made.

The material available included the semimonthly records of the Derrien tests on sheets showing the actual number made during these periods, covering about 15 months, with two summers and one winter, and showing also the number of tests for each type of

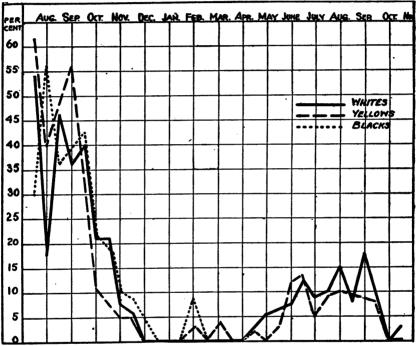


CHART 1.—(Sorgues Works) Percentage of positive tests in the Derrien examinations of urines, corrected in proportion to the numbers in each color actually tested at the time. August, 1917, to November, 1918.

employee by race and the number of clinical cases in each period which bore relation to the positive Derrien tests. It is, of course, unfortunate that in the earlier periods, in which the highest number of cases of illness occurred, the system had not been as yet evolved, so that there are no accurate records of these at present available.

As noted earlier, it is not possible to ascertain the number of employees who were actually employed at the different periods, and the best that could be done was to correct the positive tests in connection with the number of whites, yellows, and blacks whose urines were examined in the given periods. After this correction it will be seen that in general the curves for the colors approximate closely,

justifying the general impression that there is no essential difference in racial susceptibility. It will also be noted that the percentage of positive tests increases in summer and falls in winter, forming a very characteristic curve. For a variety of reasons there were rather fewer tests actually made in the winter than in the summer months, but there were enough to carry out the percentage

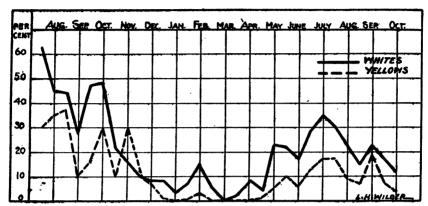


CHART 2.—(Bassen's Works) Percentage of positive tests in the Derrien examinations of urines in relation to the total number of actual chemical tests in the same period. This diagram gives the actual totals corrected to correspond with the relative number of whites and yellows actually tested at the corresponding periods.

scheme with a fair degree of accuracy. Chart I deals with the works at Sorgues, near Avignon, which were in charge of Dr. Senglars, and in which a large amount of DNP and TNP was constantly being manufactured. The two most conspicuous features are the notable difference in the numbers of positive Derriens in the two summer periods, thought to be due to the greater care in the selection of the employees and the other factors spoken of earlier in

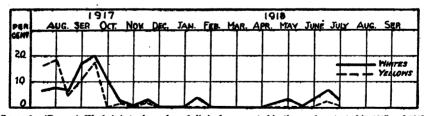


CHART 3.—(Bassen's Works) Actual number of clinical cases noted in the workers tested in 1917 and 1918. This diagram is based on actual figures for whites and yellows without regard to the relative number of each color actually employed at a given period.

this paper, and the close approximation of the curves for the three races.

Chart II is a similar series for the Bassens works, near Marseille, where the conditions were less good, and shows essentially the same points. In Chart III, also dealing with Bassens, where there were a good many clinical cases, the relative number of actual clinical

cases analyzed according to color show a marked reduction in the second year, due again to the improvement in care and in conditions of work. At these works there were practically no blacks employed, so there has been no notation of records.

II. INTOXICATION BY OTHER EXPLOSIVES IN ADDITION TO DI-NITRO-PHENOL.

The main explosives which the French have used in the course of the war have been tri-nitro-phenol and tri-nitro-toluene and combinations of these with each other and with di-nitro-phenol.

Picric acid or tri-nitro-phenol or melinite.—This was the favorite in prewar times and the main supply was obtained from Germany. so a new phenol industry had to be set up. It was therefore possible to get an idea as to the toxicity of this chemical through all the procedures of manufacturing and handling. The result may be briefly summarized as follows: With the exception of the staining of the skin of the operatives, there have been no serious results or even serious cases of sickness. In the experimental work done at Lumières in September, 1916, the dose by mouth of melinite necessary for the death of a guinea pig was taken as unity, and it was found that onefifth or less of DNP had the same effect, while the various toluenes required two or four times the dose. Through all the experiences it has not only been considered that there is no danger, but the TNP. melts have been used as resting places for the workers who have the early intoxications with DNP. The intoxications from this may therefore be dismissed as certainly of little importance.

Many of the explosives used in quantities changing according to the requests of the department of munitions have been varying mixtures of DNP, TNT, and TNP; it has been found that the toxicity had been more marked in all cases where there was most DNP, though there could be no scale established. In general, in fact, the presence of DNP in any notable amount in a mixture was about as dangerous as where it was handled alone. The degree of the danger, of course, was in direct relation to the amount accessible to absorption.

Di-nitro-toluene.—The process of manufacture results in the formation of several isomeres, which the French consider as the toxic part. These are rather oily in character and by suitable processes can be taken out, leaving a purified substance which is practically nontoxic. Perhaps the chief complication of the purification process is that the removal of the oily parts makes the material much dustier, so there is some irritation to the mucous membranes and a possibility of absorption. It is clear, however, that if the toxicity is removed by the process the dust is a nuisance rather than a danger.

During the process of removal of the oily products, the workers handling the blocks were often made ill, as were also those exposed to the vapors from the washing process. In one shop there were 25 workmen and at times half of these were incapacitated. This led to a study of the oils isomeric to the DNT and the following information was obtained:

There are four isomeres, all of which are represented in the oily substances which must be removed for the purification. Of these the 2-6, 2-5, 3-4 are the most frequent. Moreover, if the nitration has been incomplete, there is also some metal MNT found in the oil. This is the most difficult of the MNT isomeres to nitrify, so it is clear why it appears in the oil. In the work at St. Fons it was found that there was about 25 per cent of undesirable oil in the DNT, which was thrown out into the sewers without attempt at salvage, and was in fact called "Huile d'égout."

The French thus believe that it is practicable to obtain a pure DNT, with low toxicity, as a base for the final nitration.

Tri-nitro-toluene.—While this was less used among the French than among the English and Americans, there was a great deal made. at least in sufficient amount to expose many workers to its effects. The operatives were no more cleanly than the English and Americans, yet in France the experience has differed from that of the other countries. Contrary to the English idea, the French consider the manufacture of TNT to be essentially safe and have had few disabilities. There have not been recorded more than two cases of jaundice with fatal outcome, and while it is of course possible that in the hurry of war conditions there may have been some cases missed, the general statement is that in the case of deaths of the operatives, the inquiries set up by the authorities were very rigid. What is perhaps more important is that the French claim that there has been very little disability involving loss of time among the workers so that it has not been considered necessary to have change of occupation or any special medical investigations.

They consider that the purification by "sulphitization" is essential for the removal of the asymmetric isomeres, and for the removal of the tetra-nitro-methane, now known to be poisonous.

Where the purification has been properly carried out, the French consider that the toxicity is small, as was formerly considered to be the case, and are of the opinion that the troubles which have been experienced in England, and to a somewhat less extent in the United States, are due to the fact that the toxic isomeres have been left in to a greater or less extent. In this connection it may be noted that in the present process in England the sulphitization is used with some care, and that there are many fewer cases of intoxication. Moreover, where these do occur, they are less in degree than in the earlier

days. Like all statistics where there are several features to be considered, one must be cautious, but it certainly appears to be post hoc, whether propter hoc or not, as the various other methods of control have been introduced at about the same time.

In view of their disagreement, it is not out of place to bring in at this point a translation of the French process of the manufacture of tri-nitro-toluene.

Manufacture of Trinitrotoluene.

The manufacture of trinitrotoluene involves three processes of nitrification and one of purification, and the finished product should be 99 per cent symmetrical TNT of the 2-4-6 variety.

Mono-nitration.—It is necessary to use 2 to 5 per cent more HNO₃ than is required by the equation, which is as follows:

$$C_6H_5CH_3+HNO_3=C_6H_4$$
 NO_2
 H_2O

and in the process enough H₂SO₄ must be used so that the residual acid after the nitration will contain a maximum of 30 per cent water. The temperature adopted is 35° to 40° C.

The result contains a mixture of the three isomeres—ortho, meta, and para—in about the proportions of 60, 5, 35 per cent. It solidifies at $+3^{\circ}$ C. by the crystallization of para-nitro-toluene.

Di-nitration.—The nitration of this crude mono-nitro-toluene gives a mixture of isomeric di-nitro-toluene, melting at 56°, if free from mono- and tri-nitro-toluene. It contains 2-4, with a melting point of 70.5° derived from the nitration of the ortho and para mono-nitro-toluenes, and forming the greater part of the mixture. It contains also 2-6, melting at 61°, formed at the same time as the 2-4 at the expense of the ortho-mono-nitro-toluene. In addition there is 2-5, melting at 52.5°, and 3-4, melting at 60°, both of which are formed from the meta-mono-nitro-toluol, and it is these that cause the oily character of the crude stuff.

The oxidation is more intense than in the mono-nitration, with the destruction of some organic matter and with the change of some of the nitric acid into the nitrous products. It is therefore necessary to use about 10 per cent more HNO₃ than is called for in the formula:

The H₂SO₄ must be in sufficient excess to have at least 74 per cent of it left in the residuary acid.

For this di-nitration the residuary acids left after the tri-nitration are strong enough in H₂SO₄ but require the addition of HNO₄

and are employed for di-nitration of the mono-nitrate in quantities equal to those left from the tri-nitration of the amount of di-nitro-toluol desired. The temperature of the process is 50°-55°, and the ultimate heating is 90° C.

Tri-nitration.—Inasmuch as the product comes from the nitration of a mixture of isomeres, it also is a mixture as follows:

MONO-NITROS	DI-NITROS	TRI-NITROS	MELTS AT
Para	2-4	2-4-6 alpha	80.8° C
Ortho	2-6	•	
Meta	3-4	2–3–4 beta	112.0° Ć
	2-5	2-4-5 gamma	104.0° C

These are found in the proportion of about 95 per cent of the 2-4-6 and 4 to 5 per cent of the others, among which the 2-4-5 is in the largest amount. There are also small amounts of oxidation products of an acid type, and traces of tetra-nitro-methane, of which these mixtures possess the characteristic smell.

The process is accompanied by an oxidation which will destroy about 5 per cent of the product and change a notable part of the nitric acid into nitrous products. The transformation needs about 120 per cent more nitric acid than is called for in the formula, to compensate for the loss of nitric acid due to oxidation, and for the additional loss of acid due to its distillation in the course of the process.

$$\begin{array}{c} CH_{3} \\ C_{2}H_{3} \\ NO_{2} \\ NO_{2} \\ \end{array} + HNO_{3} = C_{6}H_{2} \\ \begin{array}{c} CH_{3} \\ NO_{2} \\ NO_{2} \\ NO_{3} \\ \end{array}$$

The sulpho-nitric acid used in this stage of the process must be absolutely anhydric. The amount of H₂SO₄ must be sufficient to leave 88 per cent in the residuary acid, which, as noted earlier, can then be used for the di-nitration with the addition of adequate HNO₃. The figure is not absolutely accurate on account of the abundant nitrous products whose activity is not clear. They appear, however, to reduce the activity of the bath in a manner analogous to that of water. In the process the temperature is 80° to 105° C, and the ultimate heat 120° C.

Purification.—The asymmetric isomeres must be removed, as they are oily and prevent perfect transmission of the explosion. The process is known as sulphitization.

Sodium sulphite in aqueous solution acts readily on these isomeres when diluted in a large amount of the *symmetric* forms, and changes them into di-nitro-sulphonic derivatives. The detail of the process is shown in the formula:

$$CH_3 C_6H_2 \underbrace{ \begin{array}{l} NO_2 \\ NO_2 + Na_2 SO_3 = CH_3 C_6H_2 \\ NO_2 \\ NaSO_3 \end{array}}_{NaSO_3} + NaNO_2$$

The acid oxidation products which exist in traces are also eliminated by this treatment, and the tetra-nitro-methane is also broken up, as noted elsewhere. (Appendix 4.)

To prevent attack on the *symmetric* tri-nitro-toluene the reaction must take place at a temperature low enough for this to be in the solid form. Moreover, to get a good result the sulphite must work on the crude tri-nitro-toluene when this is in a finely divided condition and preferably crystalline. By cooling the crude material while it is in active agitation in its own weight of water, it crystallizes in fine needles and is in suitable form for the reaction. This takes place best at about 25° or a little higher.

It is essential to have a crude tri-nitro-toluene free from di-nitro-toluene and from xylite in order to obtain a purified tri-nitro-toluene with a melting point above 80° C.

Thus according to the French ideas the chief stages of danger in the manufacture of the tri-nitro-toluene are in the intermediate stages when the nitration is not completed, the di-nitro-stage, on account of the oily isomeres, and in the final stages of the finished product, when the last traces of the impurities are being carried off. After this, the toxicity of the material is, in their opinion, minimal. The danger from tetra-nitro-methane is taken up in detail, and while they now acknowledge the toxicity, they claim that the sulphitization removes all this substance and that there should be no trouble from it in the finished product. (Appendix 4.)

Other nitrated bodies.—The results obtained in these various investigations led to a further series of studies of the various bodies used in the manufacture of the explosives and of the by-products resulting from the processes. In general, the two points studied were the minimum fatal doses and the relation of intoxication to the vasomotor phenomena.

Samples of a variety of materials were obtained, including-

di-nitro-anisol, tri-nitro-anisol, chloro-benzene, di-nitro-chlor-benzene, para-nitro-chlor-benzene, which were compared in the same series of experiments to-

di-nitro-phenol 2-4, tri-nitro-phenol, di-nitro-toluene 2-4, di-nitro-toluene 2-6,

as shown in the following chart taken from the article by Mayer, on the toxicity of nitrated compounds, found as Appendix 7.

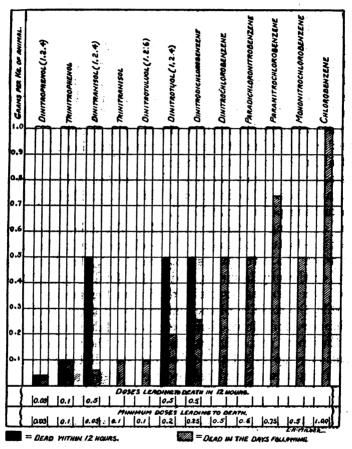


CHART 4.—Minimum fatal dose (for the dog) of different derivatives of benzene in subcutaneous injections in oil.

References.

Inasmuch as practically all of the material used in the preparation of this paper is either still in MS or is printed in a form which is not available for general distribution, and will not be accessible to the ordinary student for an indefinite time, the essentials have been translated and added to the article as appendices and references have been made to them in the text. For convenience, however, a list is appended, with the titles and the position in the appendix.

- L'expertise chimique dans les cas d'intoxication par le di-nitro-phénol. Recherche Toxicologique du Di-nitro-phenol et de l'Acide Picrique par M. le Dr. Guerbet. Paris, 1918. Ministère de l'armement et des fabrications de guerre.
- 2. Conférence pour l'étude de la Toxicité des Explosifs. Paris, 1917. Ministère de l'armement et des fabrications de guerre.

Properties of di-nitro-phénol. M. Blaise.

Toxicity of di-nitro-phenol. Dr. André Mayer.

Portals of entry. Transformation and elimination of the poison. Investigations of the derivatives in the urine, Dr. Guerbet.

Symptomatology, diagnosis, prophylaxis, treatment. Dr. Martin.

 Experimental studies on the toxic properties of di-nitro-phenol and other nitrated benzene derivatives.

Manuscript. Animal Experimentation. Dr. Mayer. An extensive and important work. Summary given in body of article.

- 4. Intoxication par le Di-nitro-toluene. Kovache. Appendix 2. MS.
- 5. Intoxication par le Tolite. Kovache. Appendix 3. MS.
- Note sur l'elimination des Tolites Brutes d'un produit toxique. Le Tetranitro-methane. Muraow. MS. Appendix 4.
- Sur les effets vasculaires de l'inhalation des Vapeurs Nitrés. André Mayer.
 MS. Appendix 5.
- Sur les effets toxiques des Huiles de Xylene et des Toluenes Nitrés. André Mayer. MS. Appendix 6.
- Sur la toxicité de quelques dérivés du Benzene. Phenol et Toluols Nitrés Industriels. André Mayer. MS. Appendix 7.
- 10. Rapport sur la Création, le Fonctionnement et les travaux de la Conférence pour l'étude de la Toxicité des Explosifs. MS. Appendix 8.

APPENDIX 1.

Reaction of Derrien.

This is the reaction used by the French in the study of the urines in di-nitro-phenol intoxications. The formation of an "azoic" by diazotation of the amino-nitro-phenols and copulation with the beta naphthol in ammoniacal solution has been used by Prof. Derrien to identify picramic acid in the urine. The "reaction of Derrien" implies the finding of an "azoic" soluble in ordinary ether and giving a violet purple color to the solution. This reaction was later extended to the investigation of other amin derivatives of the nitrophenols such as amino 2 nitro 4 phenol, and amino 4 nitro 2 phenol, which give "azoics" of various tints. The expression "Derrien's reaction" must, therefore, in this connection, be understood to refer only to the original reaction, or the general term "azoreaction" must be substituted. For the benefit of those who desire to follow the essential chemistry of the process more carefully, a translation of the explanations of Guerbet will be found in the appendix. In the application of the test the "azoreaction" is obtained by "diazoting" an amino-nitro-phenol, by "copulating" the diazoic thus obtained with an ammoniacal solution of beta naphthol, and finally by recovering in an ethereal solution the azotic coloring matter which has been formed.

DERRIEN REACTION: TECHNIQUE.

To urine recently passed	10	cc.
Add 10 per cent H ₂ SO ₄	1	cc.
Then 0.50 per cent NaNO		

shake and leave in the dark for five minutes. In another tube with a capacity of at least 25 cc. place—

Freshly made beta naphthol, 0.5 per cent solution in (22 B)	
ammonia	2	cc.

Pour the treated urine into the beta naphthol, shake, allow to stand a minute or so and add—

Ordinary ether _____ 10 cc.

Close the mouth of the tube with the finger, shake well, then cork with cork and allow the separation of the ethereal solution to take place. The color of this portion is the only one to be considered, the color of the aqueous portion having no importance.

INTERPRETATION.

If the color of the ether is violet, or wine color, or orange red, the reaction is said to be positive.

If it is colorless or yellow the reaction is said to be negative.

In an arbitrary way the degree of the reaction can be estimated in the following manner:

Make up a stock solution of-

Potassium permanganate	0. 20 gms.
Potassium bichromate	. 75 gms.
Distilled water	.000 00 gms

In 12 tubes of the same type as those used for the Derrien test, make dilutions as follows:

Stock.	Water.	Corresponds to Type—
0.5	20	ī.
.5	15	n.
.5	12	III.
1.0	17	iv.
1.0	13	v.
2.0	18	VI.
3.0	18	VII.
3.0	12	VIII.
5.0	15	IX.
7.0	14	x.
8.0	8	XI.
15.0	0	XII.
<u>, </u>		l

It is, of course, to be understood that this is not exact, but it is accurate enough to be convenient and usable.

The stock solution is very stable under ordinary precautions but the dilutions must be made up fresh for each series, as they are good only for four or five hours. The tubes for the dilutions should be washed with sulphuric acid and permanganate of potash and rinsed in distilled water.

CHEMISTRY OF AZOREACTION.

When the salt of a primary aromatic amine is treated by nitric acid (acide azoteux), a "diazoic" or "salt of diazonium" is formed. This is called "diazotation." If this salt is combined with a phenol, an "azoic" (oxyazoic) coloring matter is obtained. This reaction is a copulation, and is obtained by the use of the phenol in alkaline solution. The azoics whose formation allows the identification of the amino-nitro-phenols under discussion are obtained by copulation with beta naphthol, and are known as "azonaphtols." In reactions they act as acids with a strength depending on the number of NO, groups in the particular amino-nitro-phenol from which they started. It is the hydroxyl atom of the amino-nitro-phenol which plays the rôle of acid, and when this hydroxyl group is combined with a base—ammonia in the Derrien test—the azoic is in general very highly colored. This is notably the case in the azo-beta-naphthols corresponding to the amino 2 nitro 4 phenol and to picramic acid. When, however, the hydroxyl is set free by the action of an acid it is colorless or yellowish.

The freeing of the hydroxyl group combined with a base is the easier to obtain in direct proportion to the diminution of the NO₂ groups and the increase of the strength of the acid. Thus the azobeta naphthol which corresponds to the amino 2 nitro 4 phenol is easily liberated from its base by dilute acetic acid, while that which corresponds to picramic acid requires dilute sulphuric, acetic acid having no effect.

The azo-beta-naphthols obtained from picramic acid, from amino 2 nitro 4 phenol, and from amino 4 nitro 2 phenol, are soluble in water in the absence of ammoniacal salts, and under these conditions do not give up their coloring matter to ether. But in the presence of ammoniacal salts the azoics are precipitated in the aqueous solution and readily taken up by the ether. This is the condition which obtains in the azo reaction, and there is a tendency to have the color enhanced by the increase of the ammoniacal salts.

NOTES ON THE REACTION.

The salt of diazonium is affected by light and must be protected till ready for the second part of the reaction, and must not be heated, as it is altered by heat. The naphthol solution must be freshly made and filtered, and must be colorless. On standing there is a yellow color formed which is soluble in the ether and interferes with the accuracy of the reaction. It is important to pour the diazo solution into the naphthol solution, as if this process is reversed there may occur the formation of nitroso-beta-naphthol, which interferes with the reaction.

The delicacy of the reaction as applied to the amino-nitro-phenols is very great, mounting to one in one million, and is far more delicate than any other test which has been brought forward.

The amino 2 nitro 4 phenol and picramic acid gives azoics whose colors in ethereal solutions are very difficult to distinguish, varying in color from wine red to permanganate violet.

The amino 4 nitro 2 phenol gives an azoic whose color in the ethereal solution is a more or less orange yellow.

The authors have carried out in some detail the tests necessary for the further determination of the various compounds in the blood and organs and the differentiation in detail in the urines, but for the purposes of this paper this detail is unnecessary. It is to be hoped that the original work of Guerbet and of Mayer, now in MSS., will soon be published, as there is much detail of interest which can not be brought forward without a practically complete translation.

APPENDIX 2.

Intoxication by Di-Nitro-Toluene.

During the period of March to October, 1915, the factory of the Society for Chemical Industry at Bale, branch of Saint Fons, produced about 100 tons of drained di-nitro-toluene.

The drainage consists in enriching the di-nitro-toluene 2-4, by draining off the greater part of the oily products which accompany this isomere in the crude di-nitro-toluene.

To effect this operation the crude, washed di-nitro-toluene was poured into buckets of iron and left to solidify slowly. After which the cakes of di-nitro were broken up and left for 24 hours on a perforated stage in a room at a temperature of 25°.

We have seen several cases of intoxication caused by the washing and the draining of the di-nitro-toluene. This last operation was particularly dangerous, inasmuch as it necessitated the handling of the cakes of di-nitro-toluene. On several occasions we have had 50 per cent of our personnel (25 workmen) incapacitated.

COMPOSITION OF THE DRAINED OIL.

The oil produced by the draining of the di-nitro-toluene is a mixture of the 4 di-nitro-toluenes 2-4, 2-6, 2-5, 3-4, but much richer in the isomeres 2-6, 2-5, and 3-4 than the crude di-nitro-toluene.

If the dinitration has been imperfect and the di-nitro contains mono-nitro-toluene, the latter accumulates in the drained oil. As the meta-nitro-toluene is the most difficult isomere to nitrate of the three mono-nitro-toluenes (ortho, meta, and para), it is admitted that it predominates in the mono-nitro-toluene, is likely to exist in the imperfectly nitrated di-nitro, and therefore it passes into the waste oil supplied by this same di-nitro-toluene.

Under the conditions in which the drainage of di-nitro was practiced at the factory at Bale at St. Fons, 100 parts of crude di-nitro-tcluene (M. P. 55°, 56°) gave about 25 parts of oil and 75 parts of drained di-nitro (M. P. 62°, 63°).

ABSORPTION OF THE DI-NITRO-TOLUENE BY THE ORGANS.

It may, in our opinion, be effected in three different ways:

- 1. Through the respiratory tracts, caused by remaining in the workshops where vapors of the products exist, such as the—
- (a) Draining room—the di-nitro-toluene and its oils have a vapor tension appreciable at ordinary temperature;
- (b) Washing room—the watery vapor which escapes from the vats carries small quantities of the product.
- 2. Through the digestive tracts, caused by eating without having carefully washed the hands and nails.
- 3. Through the skin, caused by handling the product, impregnating the clothing with di-nitro, and especially the socks, which are in direct contact with the skin; and by handling tools impregnated with di-nitro-toluene (handles of shovels, etc.).

SYMPTOMS OF THE INTOXICATION.

Cyanosis: Violet blue coloration of the lips and lobes of the ears, face livid (as though seen by the light of a mercury lamp or sodium light).

Dizziness (the patient appears drunk).

Tendency to sleep, headache, dyspnæa, brown urines.

We have always seen cyanosis even before the patient felt indisposed.

Several workmen employed in the drainage of di-nitro have suffered pains in the joints, especially in the knees; these pains continued for several months after the men had left the factory at Bale and had given up the manufacture of di-nitro-toluene. One of our workmen (E. D.) suffered eye trouble (weakening of the sight), but it is possible that this man suffered from some other disease having no connection with the intoxication.

It has been noticed that some people offer more resistance than others to the action of the product. Alcoholic subjects have very little resistance.

The accidents caused by the raw di-nitro-toluene are much more frequent in summer than in winter. We have never had any fatal accidents; the greater number of patients recovered after two or three days, and much more rapidly if they left work as soon as the first symptoms appeared.

MEASURES TAKEN TO COMBAT THE INTOXICATION.

General measures: As soon as cyanosis appears have the workman leave the workroom and walk in the open air.

Fight the tendency to sleep by having workman drink coffee.

Ventilate the workroom.

Give no alcohol-no wine-give milk.

Avoid touching the product with the hands. Our workmen have always worn rubber gloves, but these become dangerous if the product penetrates to the inside. This often happens, in which case the skin of the hands fairly macerates with the noxious substance.

Attention to the personal cleanliness of the workmen: Washing the hands with carbonate of soda before leaving the workroom at meal times.

No eating in workroom.

Special clothes for working.

Outdoor clothes and knapsacks containing food should be hung in the cloakroom and not in a corner of the workroom.

St. Chamas, May 27, 1918. Military Technical Agent. Signed: KOVACHE.

APPENDIX 3.

Intexication by TNT.

During the stay of a year at the factory of Neuville (1916) we have to report the death of only one workman affected by the grinding of the product; but the autopsy proved that this man had been alcoholic (cirrhosis of the liver).

The male and the female workers affected by the grinding of crude TNT show a livid tint of the face, but never complain of pains.

The grinding of TNT makes no perceptible dust, the product having a greasy consistency, but, on the other hand, it is made unpleasant by the presence of tetra-nitro-methane, which irritates the eyes. This substance is given off by the crude TNT during grinding, which increases the surface of the product exposed to the air. It has an appreciable vapor tension, for TNT, ground a certain time and exposed to the air, no longer smells, or irritates the eyes.

It seems that in the American factories the odor of crude TNT and its tear-producing properties have seriously discommoded the workers, because, ever since November, 1915, it is known that the Americans wash the TNT with sulphite and bicarbonate of soda for the sole purpose of eliminating the bad odor which pervades the workrooms.

It is worth while noting here that the irritating odor of crude TNT is distinctly noticed during the washing of the product with hot water (carrying off by steam).

As to the danger of tetra-nitro-methane we do not believe it to be very marked, because, according to the French patent No. 384079, of November 16, 1907, Paul Winaud proposes the use of a mixture of tetra-nitro-methane and combustible bodies to replace panclastites (a mixture of liquified nitrous gases and combustible bodies), making a point of the fact that the tetra-nitro-methane is much less toxic than nitrous vapors.

The grinding of pure TNT does not irritate the eyes but does produce dust. The purification of TNT makes it lose both its odor and its greasy consistency.

St. Chamas, May 27, 1918. The Military Technical Agent. Signed: KOVACHE.

APPENDIX 4.

Note on the Elimination From Crude TNT of a Poisonous Product— Tetra-Nitro-Methane.

A number of fatal cases of intoxication have recently been observed in England among workers manipulating crude TNT.

Moreover, in an article appearing in a German review 1 on the toxicity of nitrogen derivatives, the accidents observed in the manufacture of TNT have been attributed principally to the action of tetra-nitro-methane of which the toxicity appears to be remarkable.

The elimination of this product from the crude TNT thus appears most desirable, and we have been led to find out how the tetra-nitromethane behaves in the course of purification by sulphitization.

¹ Extraîts des périodiques Chimiques Allemands (Ministère de l'Armement, Fascicule VIII. P. 578.).

We have heretofore studied the solubility of this product and new experiments have confirmed our observations. Tetra-nitro-methane in contact, when cold, with a dilute solution of sulphite (4 to 5 per cent of anhydrous sulphite) dissolves with great rapidity and with an elevation of temperature. The reaction is much more energetic than that of asymetric TNT, and it is certain that, in the course of the purification by sulphitization, the tetra-nitro-methane disappears very rapidly. What is the mechanism of this reaction?

The most plausible hypothesis consists in admitting a reaction analogous to that of the sulphite of sodium on asymetric TNT, namely, the substitution of a sulphite group for a nitrite group.

By the reaction:

$$C(NO_2)_4+SO_3Na_2=C(NO_2)_3SO_3Na+NaNO_2$$

there is thus obtained tri-nitro-methane sulphite of sodium soluble in water.

In fact, the reaction is more complex, since the preceding equation implies a maintenance of neutrality, whereas the solution becomes acid very quickly and there is observed a release of nitrogen dioxide.

This phenomenon appears to us to be explained in the following manner:

As a first phase there is indeed formed tri-nitro-methane sulphite of sodium, but this unstable compound hydrolyzes quickly, according to the equation:

$$C \left(\frac{(NO_2)_3}{SO_2Na} + H_2O = C \left(\frac{(NO_2)_3}{H} + SO_4NaH\right)\right)$$

giving rise to tri-nitro-methane and sodium bisulphate.1

The reaction of these products on the sodium nitrite formed in the first reaction would give rise to nitrogen dioxide.

In order to confirm this hypothesis we have tried to identify the tri-nitro-methane formed.

The yellow solutions obtained present the characteristics of this body: Discoloration under the action of very concentrated acids and the passing over of the coloring body in the steam. To identify the tri-nitro-methane we have proceeded in the following manner:

Three cc. of tetra-nitro-methane are treated with a solution of 10 gr. of sodium sulphite in 100 cc. of water. The solution, of a strong yellow color, is acidified by H₂SO₄, then extracted with ether. The ether is treated with an excess of silver oxide to a neutral reaction; the ether solution is then decanted and evaporated.

¹ It is possible that this reaction may be due to an autocatalysis, the first traces of acid form favoring the hydrolysis of tri-nitro-methane sulphite. Perhaps the yellow solution obtained in carrying out the reaction of an ammoniacal solution of sulphite on tetro-nitro-methane may contain, unaltered, tri-nitro-methane sulphite of sodium. This point remains to be determined.

We have thus obtained the explosive silver salt of tri-nitro-methane described by Hantzoch and Hichenberger (Ber. 32 P. 636, 1899), a silver salt of which the solubility in ether may be considered as characteristic.

RÉSUMÉ.

- 1. Tetra-nitro-methane may easily be eliminated from crude TNT by the treatment of sulphitization.
- 2. The reaction seems to be carried out in two phases: (a) The formation of tri-nitro-methane sulphite of sodium, and (b) the hydrolysis of this product with the formation of sodium salt and tri-nitro-methane.

Paris, August 7, 1917. Chief Chemical Engineer. Signed: MURAOW.

Approved:

Chief Engineer in charge of the Service of Chemical Studies.

Signed: MARQUEYROL.

Approved:

Chief Engineer, assistant to the Director of the Service of I. E. E. T. P.

Signed: LIOUVILLE.

APPENDIX 5.

Note on the Vascular Effects of the Inhalation of the Vapors of Nitrated Toluene.

(Dr. MAYER.)

In a former note we have shown that when the products which leave the oils of toluene, maintained at 100° C., are carried off in steam, these products inhaled by an animal produce no vasomotor effects.

It is desirable to know if the vapors given off under the same conditions by mono-nitro-toluene (ortho- and para-) or by di-nitro-toluene 1-2-4 produce vasomotor effects.

We have made experiments to determine this question. A rabbit was tracheotomized. A canula was placed in the trachea and connected with a T-tube whose horizontal branch was traversed by an air current charged with the vapors given off when the nitrated toluenes were heated to 100° C. in a hot-water bath. The blood pressure and pulse were registered. The inhalation continued for about 15 minutes.

It can be seen from the attached curves that this inhalation produced no vascular effects.

Note.—The curves noted, which can not be readily reproduced, show that there is no change of blood pressure either at the beginning or the end of the experiment.

APPENDIX 6.

Note on the Toxic Effects of Oils of Xylene and Nitrated Toluene.

(Dr. MAYER.)

It is desirable to determine whether the commercial products which result from the nitration of the oils of xylene and toluene produce toxic effects. It is especially desired to know if they have an action analogous to that of nitroglycerin, which, when introduced into the organism, gives rise to widespread vaso-dilatations.

To determine this question we have been provided with the following samples:

- 1. Oil of trinitrated xylite; Marqueyrol process.
- 2. Oil of di-nitro-xylene coming from the drying out of crude xylite, sent by the powder works of St. Chamas.
- 3. Oil of di-nitro-toluene also sent by the powder works of St. Chamas.

I. GENERAL TOXICITY.

We have undertaken to determine the toxicity of these products by injecting them subcutaneously into dogs. To make possible this injection we have put these products into suspension in oil.

The results of our experiments are condensed in the following table:

Dose in	Oil of di-nitro-toluene.		Oil of di-nitro-xylene.		Oil of xylite trinitrated.	
per kil- ogram of ani- mal.	Weight of dog.	Result.	Weight of dog.	Result.	Weight of dog.	Result.
	Kg.		Kg.		Kg.	
0.5	9.	Dead after 20 hours	12	Dead after 24 hours	12	Dead after 24 hours
.2	10.5	Survived	9	Survived	21	Dead after 30 hours.
. 1	15	đo	14	đo	32	Survived.
.1					. 15	Do.
. 05	16.5	Survived	14.5	Survived	18	Do.

It is seen that the oil of di-nitro-toluene causes a fatal intoxication in doses of 0.50 gr. per kg.; the oil of di-nitro-xylene in doses of 0.50 gr. per kg.; and the oil of tri-nitro-xylene in doses of 0.20 gr. per kg. These products are thus relatively slightly toxic. The nitrated compounds of xylene and toluene appear to have the same order of toxicity.

II. LESIONS PRODUCED BY OILS OF XYLENE AND NITRATED TOLUENE.

The oils of xylene and nitrated toluene injected into animals produce toxic effects. These effects seem to react upon the principal vital organs, in particular upon the liver and kidneys.

We have studied, from a cytological point of view, the organs of animals which have succumbed after injections of the products experimented upon, and have made the following observations:

Oil of trinitrated xylite—(a) Liver.—As a whole the liver is congested. Cytologically it presents extremely definite lesions. These lesions consist in alterations of the chondriome of the type of homogenization. Almost all the areas examined in section show this homogenization in the second degree, and sometimes in the third degree. Moreover, there is noted in almost all the cells the presence of fine fatty granulations. The toxic processes thus concern the phosphatides of the liver.

(b) Kidneys.—The kidneys also present cytologic lesions; the Strands of Heidenheim have disappeared; the protoplasm is gathered together in masses in the form of a coarse network. This network holds granulations whose dimensions are sometimes considerable. The appearance of the chondriome is that of homogenization in the first and second degree.

Oil of dinitrotoluene and of dinitroxylene—(a) Liver.—The liver is congested and cytologically injured. Above all the chondriosome appears to be altered. The granulations are often indistinct. Furthermore the cells contain abnormal inclusions, which reduce osmic acid, and are soluble in the solvents for fatty bodies, thereafter leaving their trace in permanent slides in the form of round vacuoles.

(b) Kidneys.—The kidneys present slight cytological lesions; homogenization in the first degree.

These cytological observations are corroborated by chemical analysis of the tissues. The liver contains a large quantity of fixed fatty acids, and a quantity of insaponifiable substances much greater than normal (for 100 gr. dry: Fixed fatty acid, 10.89 gr.; cholesterine, 0.55 gr.; other insaponifiable substances, 2.46 gr.).

Thus the products examined, when administered in fatal doses, cause very important lesions of the liver and alterations of the kidneys.

III. THE ACTION ON BLOOD PRESSURE.

It is known that nitroglycerin (used in France under the trade name of trinitrine) is a strong vasodilatator. The vasodilatation which it produces is so extensive that the organism is not able to compensate for it, and the arterial pressure is markedly lower after administration of this product. This lowering of pressure is produced after intravenous injections in the rabbit in doses of about 0.003) gram per kilogram.

We have injected either under the skin or into the peritoneum (it has been impossible to make satisfactory injections into the veins) considerable doses of oils of xylene and of toluene. We can say im-

mediately that the effect obtained is in nowise comparable with that of nitroglycerin.

1. When these products are injected into the peritoneum there is indeed, at the moment of injection, a sharp falling of pressure. But this is to be ascribed to the puncture of the skin and the injection itself. This action can be reproduced by simple injection of oil. Shortly after the injection the pressure comes back to normal (fig. 2).

When these products are injected under the skin the fall of pressure is scarcely noticeable and is extremely brief.

2. When one follows the animal into which oils of xylene and toluene have been injected, he sees the pressure remount after injection and maintain itself for sometime at normal level. Then it lowers progressively during the time in which the intoxication progresses. There is no effect comparable to the sudden and transient action of nitroglycerin.

From a practical point of view the question comes up to determine whether the vapors given off by the oils tested may produce effects of vasodilatation.

We have undertaken to make vapors coming from these products penetrate into the respiratory tracts of the rabbit. The oil was kept boiling in a flask placed in a water bath. A current of air carried away the vapors whose odor was clearly perceptible. Next the vertical branch of a tracheal canula of T-shape was placed in the trachea of the animal and the current of air charged with vapors was sent through the horizontal branch. The rabbit thus breathed air containing the products to be experimented with.

Under these conditions we have not discovered any fall of arterial pressure in the subject under experimentation.

The oils studied do not thus emit vasodilating vapors.

CONCLUSIONS.

- 1. The oils of di-nitro-toluene and of di-nitro-xylene and the oils of trinitrated xylite are toxic. The fatal dose for the dog was for the first two, 0.50 gram per kg.; for the last one, 0.20 gram per kg.
 - 2. They produce important lesions of the liver and kidneys.
- 3. They have no vasodilating effects analogous to those of nitro-glycerine.
- 4. Their vapors, when inhaled, do not lead to a fall of arterial pressure.

NOTE.—The curves with the original show that while controls with tri-nitrine give a marked change in pressure, with the materials under experiment there are no such changes.

APPENDIX 7.

Note on the Toxicity of Several Commercial Nitrated Products of Benzene, Phenols, and Toluols.

(Dr. MAYER.)

We have attempted to establish the minimum fatal doses for several commercial nitrated products of benzene, phenols, anisols, and toluols.

- 1. Chlorobenzene coming from the liquid-air factory at Montereau.
- 2. Mono-nitro-chlorobenzene coming from Montereau.
- (a) Intimate mixture of para and ortho mono-nitro-chlorobenzene, which solidifies at about 12° C. and separates by centrifugation.
- (b) Para, perhaps containing a little of the ortho separated by being carried off by steam, and melting at about 80° C.
- (c) Para mono-nitro-chlorobenzene, practically pure and melting at about 83° C.
 - 3. Para-di-chloro-nitrobenzene, coming from Montereau.
- 4. Di-nitro-chlorobenzene, coming from Montereau, and forming, at ordinary temperatures, a mixture of oils and crystals.
 - , 5. Di-nitro-di-chlorobenzene, coming from Montereau.
 - 6. Commercial di-nitro-phenol 1.2.4, coming from Montereau.
 - 7. Tri-nitro-phenol, coming from the Gillet factory.
 - 8. Di-nitranisol 1.2.4, coming from Montereau.
 - 9. Tri-nitranicol, coming from Montereau.
 - 10. Di-nitro-toluol 1.2.4, acquired at Poulenc.
 - 11. Di-nitro-toluol 1.2.6, acquired at Poulenc.

All these bodies have been experimented upon in the following manner:

Solution in ether; mixture of the ether solution with olive oil; evaporation in electric sand bath; subcutaneous injection of the material in the oil. The experiments have been carried out on the dog. Under these conditions we have obtained the results shown in the following tables:

CHLORO-BENZENE.

Number of grams per kg. of animal.	Initial weight of the animal in kgs.	Fate of the animal.
1.0	16. 7	Survived.

MONO-NITRO-CHLORO-BENZENES.

(1. Mono-nitro-chloro-benzine. Intimate mixture of para and ortho, solidifying at about 12° C., separated by centrifuging of the first.)

ived. Do.

MONO-NITRO-CHLORO-BENZENES-Continued.

(2. Mono-nitro-chloro-benzene. Para, perhaps containing a little ortho, separated by being carried off by steam.)

Number of grams per kg. of animal.	Initial weight of the animal in kgs.	Fate of the animal.
0.5	13.5	Dead after 2 days.
.25	9	Survived.

(3. Mono-nitro-chloro-benzene. Para, practically pure.)

0.5 .5 .5	9 10 12	Dead after 24 hours. Do. Survived.
. 25	11	Dead after 5 days.

DI-NITRO-CHLORO-BENZENE.

1. OILY PART.

2. CRYSTALS.

		1	
0.3	25	Dead after 2 days.	
.3	16.5	Survived.	
.1	23	Do.	
.1	16.5	Do.	
1 1		1	

PARA-DI-CHLORO-NITRO-BENZENE.

1.0 .5 .25	7 6 6	Died during the night. Dead after 48 hours. Survived.
.1	6	Do.

DI-NITRO-DI-CHLORO-BENZENE.

1.0	58	Died during the night.
.5	7	Do.
. 25	8	Dead after 5 days.
.1	7	Survived.

DI-NITRO-TOLUOL.

1. DI-NITRO-TOLUOL 1-2-4.

0.5	6 8.5	Dead after 8 hours. Dead after 4 days.
.1	13.5	Survived.
.1	8.5	Do.
. 05	13.5	Do.

DI-NITRO-TOLUOL-Continued.

2. DI-NITRO-TOLUOL 1-2-6.

Number of grams per kg. of animal.	Initial weight of the animal in kgs.	Fate of the animal.
0.1 .1 .05	14.50 13 13	Dead after 2 days. Dead after 8 days. Survived.

DI-NITRANISOL 1-2-4.

1.0	8	Died during the night.
.5	5	Do.
.1	6	Dead after 48 hours.
. 05	7.5	Dead after 3 days.
. 05	7	Survived.
.03	7	Do.
.03	9	Do.
1	i	

TRI-NITRANISOL.

		1
1.0	6	Dead after 18 hours.
.5	6	Do.
.1	8	Dead after 24 hours.
. 05	10	Survived.
. 05	9	Do.
		1

TRI-NITRO-PHENOL 1-2-4-6.

1		1	_
0.5	4	Dead after 4 hours.	
.1	22	Dead after 6 hours.	
. 05	13	Survived.	
.05	22	Do.	
.03	13.8	Do.	
.03	13.6	Do.	
1 1			

DI-NITRO-PHENOL 1-2-4.

0.1	22.7	Dead after 1 hour.
.1	17	Do.
.05	20	Do.
.05	13.5	Deadafter1hourand30minutes.
.03	22	Dead after 2½ hours.
.03	13	Dead after 3 hours.
.03	12	Survived.
.02	17.5	Do.
.02	17	Do.
]		

We have condensed our results into a graph (see Chart 4) which indicates the minimum fatal dose either for 12 hours or for 10 days following the injection.

The inspection of this graph shows that certain bodies in the minimum fatal dose give rise to a superacute intoxication. Such bodies are di-nitro-phenol 1-2-4 and tri-nitro-phenol. Other bodies, on the contrary, when injected in limited doses, produce only a slow intoxication.

We may add that among these latter bodies certain of them are able, if injected in sufficiently strong doses, to lead rapidly to death, while others, even in strong doses (1 gram per kg.), do not produce death in 12 hours.

CONCLUSIONS.

- 1. The chloro and nitro-chloro-benzene are clearly less toxic than the nitro-toluol, anisols, and phenols.
- 2. For the commercial products which we have had in hand the doses leading to death in 12 hours are as follows:

	m per animal.
For the di-nitro-phenol 1-2-4	0.03
For the tri-nitro-phenol	. 10
For the di-nitranisol	. 50
For the di-nitro-toluol 1-2-4	. 50
For the di-nitro-di-chloro-benzene	. 50

The minimum doses leading to death are as follows:

Gra	m per
kg. of	animal.
For the di-nitro-phenol 1-2-4	0.03
For the di-nitranisol 1-2-4	. 05
For the tri-nitro-phenol	. 10
For the tri-nitranisol	. 10
For the di-nitro-toluol 1-2-6	. 10
For the di-nitro-toluol 1-2-4	. 20
For the di-nitro-di-chloro-benzene	.25
For the di-nitro-chloro-benzene	. 50
For the para-di-chloro-nitro-benzene	. 50
For the para-nitro-chloro-benzene	¹ . 75
For the mono-nitro-chloro-benzene	. 50
For the chloro-benzene	1.00

2375 October 24, 1919.

DETERMINATION OF BACTERITROPIC CONTENT OF ANTIMENINGOCOCCIC SERUM.

By ALICE C. EVANS, Bacteriologist, United States Public Health Service.

There are several possible methods for testing antimeningococcus serum for its content in immune antibodies, but none of them is considered entirely satisfactory. The agglutination reaction is the simplest of these tests and it is the one most commonly applied. Inasmuch as the part which agglutinins may play in the therapeutic activity of the serum is unknown, a high content of agglutinins in a serum does not give assurance that the serum in question would be efficacious in the treatment of disease. Investigators of serum immunized against various kinds of pathogenic organisms have shown that a high agglutinin content is not always paralleled with a high content of other known antibodies.

The complement fixation reaction is also commonly used for testing antimening occicic serum, but this test is not entirely satisfactory for a similar reason, and also on account of the great complexity of the test. Neither the phenomenon of agglutination, nor that of fixation of complement (since complement is lacking in the cerebrospinal fluid) appears to be a necessary part of the human defense in cerebrospinal meningitis.

Judged by the picture of the disease, it appears that those antibodies which act upon the invading organisms in such a manner that they are prepared for ingestion by the leucocytes probably play an important part in the therapeutic activity of the serum. If that is true, then a quantitative determination of the antibodies promoting phagocytosis should give some indication of the therapeutic value of a serum. Heretofore a reliable method for determining the phagocytic antibodies has not been described, and the "opsonic" test, as it was called, was rejected because experienced laboratory workers could not obtain consistent results.

In view of the importance of a reliable test by which to judge the large quantities of antimeningococcus serum needed to combat the outbreaks of miningitis in the army camps, a study of the phagocytosis-promoting bodies of antimeningococcic serum was undertaken as a war emergency problem. Before a satisfactory method for carrying out this test had been developed, the signing of the armistice relieved the urgency for haste in perfecting a new method; consequently there was undertaken a more comprehensive study of the meningococci in our collection than was at first contemplated.

It was found that the important phagocytic antibodies of antimeningococcic serum are not the labile opsonins which depend upon complement for their activity, but they are the more stable bacteriotropins. Contrary to the report of Kolmer (1)¹ and his associates, who recommended the addition of complement to antimeningococcus

¹ Number indicates reference citéd.

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serum before using it for treatment, there was never observed any reactivating effect when complement was added to commercial serum.

The early investigators of meningococcus serum reported that some of their strains were resistant to phagocytosis, and would not respond to treatment with immune serum. They attributed such a resistance to the inherent properties of certain strains. But it is now evident that the strains were found to be resistant because those investigators expected a serum immunized against one strain of meningococcus to react with every other strain of meningococcus, whereas it will react with only such strains as belong to the same specific group.

Dopter (2)¹ found two groups of meningococci distinguishable by their agglutinin reactions. Gordon (3)¹ also studied the agglutinin reactions of meningococci and grouped the epidemic strains in England into four types.

Similarly, I have found distinct groups of meningococci distinguishable by their tropin relationship. Four such groups were distinguishable which have been designated R, S, T, and U. A fifth group, Z, is made up of organisms which are more or less closely related to all the other groups.

To some extent there is a coincidence between the agglutinin types and the tropin groups. Group R includes the majority of strains of Gordon's Type I. It also includes the majority of strains of Type III and a few strains of other types. Group S includes the majority of strains of Type II, and a few strains of other types. Group T is a small group made up of strains of various types. Group U was represented by a single strain in our collection.

Groups R, S, T, and U are well defined. Only a few strains belonging to these groups were found to have any tropin relationship with the other groups. For example, a strain of Group S will show no reaction in a Group R serum. Obviously, so long as these groups were unrecognized in phagocytic studies and a serum obtained by immunizing against one strain of meningococcus was expected to react with every other strain of meningococcus, inconsistent results were inevitably reported.

No strain of meningococcus was found to be resistant to phagocytosis after treatment with specific tropins, and every tested strain was found to possess tropinogenic powers. But those unqualified statements do not apply to all strains which have been maintained for a long time under artificial conditions. Strains in our collection have been observed to weaken in their tropinogenic powers, and also in their power of response to specific tropins. The entire loss of both properties has been observed in a few strains.

There is a practical significance in the loss of antibody producing properties by strains of meningococci after long artificial cultivation.

Such strains can not be relied upon as antigens for the production of serum for therapeutic purposes.

It has been the common observation of investigators that the agglutinin content of immune serum is not always paralleled by the tropin content. That was found to be the case with antimeningo-coccic serum. In the majority of serums prepared for experimental purposes a good production of agglutinins was found to be accompanied by a good production of tropins; but strains which had lost their tropinogenic power were shown to possess good agglutinogenic properties. On the other hand, strains with a poor agglutinogenic power were found to produce serum with a good tropin content.

The two antibodies are affected differently by adverse conditions. The agglutinins were destroyed at a lower temperature than the tropins; but some other adverse conditions were found to destroy the tropins more readily than the agglutinins.

Thus it was shown that the two antibodies are independent of one another in their characteristic effects on meningococci. It is therefore not to be expected that a commercial serum containing a good content of agglutinins will always contain a good content of tropins.

One hundred and twenty-eight commercial serums have been examined for their content of tropins specific for Groups R and S, and the results were compared with the agglutination reactions of those serums as determined at the Hygienic Laboratory.

The two tests agreed in the case of about 70 per cent of the serums, but for the remaining serums the results were discordant; so that a serum which was rejected by the agglutinin test would have passed if judged by the tropin test alone, or vice versa. In the case of some of the serums for which the results were discordant the content of both antibodies was near the lower limit—that of the one lying just above and that of the other lying just below the arbitrary standard. But a few of the serums showed a high content of the one antibody and a low content of the other.

The tropin test has proved to be a workable test, giving results as consistent as can be expected in such a complicated biological reaction. Retests on the same serum have shown practically the same content of tropins for the various groups of meningococci as in the original tests.

The full report, including tables giving the data upon which the above conclusions were based, is now in manuscript, and will be published in a Hygienic Laboratory bulletin.

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THE DIAGNOSIS OF RABIES IN ANIMALS.

A STATISTICAL STUDY OF THE RECORDS OF THE HYGIENIC LABORATORY FOR THE PERIOD 1909 TO 1919.

By H. E. HASSELTINE, Passed Assistant Surgeon United States Public Health Service.

This study, from the records of the Hygienic Laboratory, was undertaken to determine the reliability of the microscopical examination for the presence of Negri bodies as a means of diagnosing rabies. From February, 1909, to April 30, 1919, 1,127 specimens from suspected animals of various species have been submitted. While the examinations have been made by a number of officers of the Public Health Service, the general technique followed has been practically constant throughout.

Technique of tests.

A description of the technique employed will serve to make clear the different types of examination. Three types of tests have been used which may be designated as the smear, the section, and the inoculation tests. Some brains were subjected to all three tests, but after a time this was discontinued as it was apparent that when Negri bodies were found by microscopic examination the inoculation test was almost invariably positive. Consequently the incculation test was used only in case the microscopic examination failed to show Negri bodies, or in cases in which the bodies found were of doubtful character.

The smear test, advocated by Williams and Lowden, is performed as follows: A thin section of the hippocampus major (Ammon's horn) of the brain of the suspected animal is removed and placed on a clean glass slide. It is then crushed between two slides and the slides drawn apart making a uniform smear of the brain tissue upon both slides. The smear is flooded with methyl alcohol (ethyl alcohol may be used) for one minute. This is poured off and the smear is stained with fuchsin-methylene blue, the preparation being steamed slightly for about one minute. The stain used by the Hygienic Laboratory is prepared fresh each day as follows: To about 50 c. c. of distilled water add 3 drops of saturated alcoholic solution of basic fuchsin and about 5 drops of saturated aqueous solution of methylene blue. Mix well and use at once. If the smear is too red, or too blue, the stain may be modified by adding 1 or 2 more drops of the stain desired and repeating the staining process on another smear.

After the smear has dried it is examined as follows: With the low-power lens a field showing large pyramidal cells of the hippocampus major is located. The field is then covered with a drop of oil and examined with the immersion lens. The large cells should be stained blue; the Negri bodies, if present, are stained red and found lying

within, or near, the cells. Closer examination usually reveals one or more small blue dots within the Negri bodies, though at times these are difficult to demonstrate.

To make a positive diagnosis Negri bodies should be found within the cells, though the method of preparation often results in bodies being found outside the cells. Owing to the resemblance of red blood cells to the extracellular bodies, the finding of intracellular bodies is the criterion of a positive diagnosis.

If the smear gives a positive result, no further test is made. If negative, a section of the hippocampus major may be taken and placed in fixing solution for the preparation of sections. If Negri bodies are not demonstrated by the smear method, a portion of the brain (hippocampus, medulla, or other part) is placed in glycerin for about three days, to be used for the inoculation of animals.

Before recording a negative finding by the smear method, several smears should be stained and examined, and they should be made from the hippocampus major of both cerebral hemispheres. In a few instances Negri bodies have been found easily in one hippocampus when smears from the opposite side have failed to reveal their presence.

The section method is a little more accurate than the smear method but the longer time required and the greater amount of work necessary in that method make the smear method the more practicable. In a few cases the section test has been the only one possible, as the brains were submitted after having been placed in formaldehyde or other fixing solution.

For demonstrating Negri bodies in sections we have found that Zenker's solution as the fixing fluid and the eosin-methylene blue stain give the best results. We have used paraffin for embedding and cutting sections. Using this technique the cells of the hippocampus are stained blue and the Negri bodies red with the blue inner bodies usually showing. All the bodies should be within the cells as the method of preparation should not break up the cells.

The animal inoculation method is used in all cases which give a negative microscopic finding. A piece of the brain tissue of the suspected animal is placed in glycerin for from three to five days. It has been found that this treatment serves to lessen the danger of abscess or other lesion which may result from contaminating organisms. Animals inoculated with a brain emulsion made without this preliminary period in glycerin frequently die in the first 3 days, a period far too short to be of any value in the diagnosis of rabies.

After the suspected brain tissue has been in glycerin for from 3 to 5 days, a thick emulsion of the tissue is made by grinding it in a sterilized mortar and adding sterilized salt solution. A small

amount, about 0.2 c. c., of this emulsion is then injected with a sterile syringe subdurally, or intracerebrally, into guinea pigs or rabbits. The animal is etherized, the skin across the parieto-occipital region is incised, and a small hole is punched in the cranium with the center pin of a trephine. The needle is passed through this hole and the desired amount injected. After the operation a collodion dressing may be applied to the skin incision if desired. At least 2 animals are inoculated from each suspected case. The animals are then observed for symptoms of rabies, and if they die after a period of 5 days an examination of their brain for Negri bodies is made. Animals should be kept under observation for at least 6 months; though most of those developing rabies will develop symptoms within 2 months.

Origin and Distribution of Specimens.

While the specimens examined came from different parts of the United States, a large majority came from the District of Columbia, Maryland, and Virginia. A greater number of specimens from the District of Columbia were examined by the Bureau of Animal Industry, Department of Agriculture, and many specimens from Maryland were examined by the health department of that State. While the figures given in the following table do not indicate the true provalence of rabies in any State, the geographic origin of specimens by States is shown.

			**	
State.	Total.	Positive.	Negative.	Not examined.
Arizona District of Columbia. Florida. Kentucky Maryland Mississippi Nebraska. North Carolina. New Mexico. New Mexico. New York Oregon. Pennsylvania. South Dakota. Tennessee Virginia. West Virginia. West Virginia. United States Army United States Army United States Army	3 7 42 1 1 2 4 2	1 1 1 1 543 5	2 16 1 3 15 1 1 1 4 1 1 360 6	33115
Total	1, 127	585	415	124

TABLE I .- The geographic origin of specimens, by States.

Most of the specimens submitted from distant States were shipped in some preserving medium, such as glycerin, formaldehyde, or other reagent.

Table II.—Distribution of positive and negative specimens among different species of animals.

Animal.	Total.	Positive.	Neg dive.	Not examined.
Dogs. Cats. Cattle Horses	922 103 52 5	514 34 32 1	306 54 18 4	102 15 2
Mules Swine Sheep Skunks	5	5 1	15 1 5	i
Squirrels Rabbits Opossum Rat	3 2 1 1		3 2 1 1	
Man Unknown	2 4	1	1	1
Total	1, 127	588	415	124

It is evident from Table II and from the natural proclivities of the animal to bite, that the dog (514 positive specimens out of a total of 588, is the greatest disseminator of rabies. It is also evident that the number of cattle (32), horses (1), and hogs (5) lost from the disease represents quite an economic loss. As most of the domestic animals represented, other than dogs and cats, do not often bite, it is probable that practically all the horses, cattle, sheep, and swine receive their infection as a result of being bitten by rabid dogs.

The seasonal prevalence of the disease in animals, as indicated by the results of this series, is shown in Table III.

TABLE III.—Seasonal prevalence of rabies in animals.

	Total for year.	ខននដ	118 110 157	<u>%</u>	1,127
<u> </u>	Not examined.		-6	- :	4
December	Negative.		8040	-61	8
Dec	Positive.	441-	~ ∞5∞	-104	55
i.	Not examined.			-	60
November.	Negative.	64	mm M	44	8
Nov	Positive.	22.91	○ 8504	7-60	22
٠	Not examined.			-8	9
October.	Negative.	-00	0000	е :	23
ő	Positive.	80 ED 89	4001	44	88
ē.	Not examined.	-120	-0-0	-60	17
September.	Negative.	8	4000	-2	ឌ
Sep	Positive.	H 44	4400	6 8	33
ند	Not examined.	-4	700	7	24
August.	Negative.	9	210010	& r3	37
₹	Positive.	1 3 3	4918	∞ ल	ន
	Not examined.	- : : : -	400	ကတ	13
July.	Negative.	3 12	8 11 5	P-01	\$
	Positive.	1 1 9	2044	10 4	37
	Not exammed.	1 2	88	-8	21
June.	Negative.	4 60	4 II 6 8	က်ဆ	\$
	Positive.	: : : : : : : : : : : : : : : : : :	F490	ဗဗ	జ
	Not examined.	*		87	=
May.	Legative.		u~+ü	∞∞	7
	Positive.	်းဂတ	∞c₁4⇔	69	46
	Not examined.	7	::~-	- 7	6
April	Negative.		r04r0	11 4 9	\$
	Positive.	-615	6664	1200	23
ند	Not examined.	63	<u>- : : : : : : : : : : : : : : : : : : :</u>	-73	စ
March	Negative.	- : : : : -	145.11	0.4 ·	8
24	Positive.		6626	752	25
ry.	Not examined.	- : :	::::	- ; ;	~
February	Negative.		0000	4410	8
	Positive.	25-2	9461	5r.8	8
y.	Not examined.	<u> </u>	64	- :-	ю.
January.	Negative.		L 524	6	8
Ja	Positive.	8	ကလတ	552	ន
	Year.	1909. 1910. 1911.	1913. 1914. 1915.	1917. 1918. 1919.	Total.

From Table III it will be seen that the greater number of positive brains were obtained in the colder months, which is directly opposed to the popular belief that rabies is more prevalent in hot months.

Table IV shows the different months of the year arranged in the order of the highest average number of positive cases.

Month.	Positive.	Negative.	Not examined.	Monthly average, positive.	Percentage positive of those examined.
March January December February November April May June October July September August	55 61 54 52 46 39 38 37	35 26 20 36 26 48 47 46 22 49 23	6 5 4 2 2 3 9 11 12 6 25 17 24	6. 87 5. 90 5. 50 5. 56 5. 40 4. 73 4. 60 3. 90 3. 80 3. 70 3. 60	68.2 72.0 73.3 62.1 67.5 52.0 49.5 46.4 63.3 43.0 61.0

TABLE IV .- Summary of Table III showing occurrence of rabies by months.

A factor which may explain the apparent lower prevalence in summer is the popular belief that dogs become rabid only in warm weather. As a result of this belief, if any dog appears sick or abnormal in warm weather the animal is promptly killed and the head sent in for examination. Consequently, in warm weather some dogs that are not rabid are killed and submitted for examination. On the other hand, in cold weather the animal is not regarded with as great suspicion, and animals not rabid are kept until they recover, while those suffering from rabies die of the disease and the brains are then submitted for examination. Moreover, it will be noted that many specimens were not examined during the warm months; but it is reasonable to suppose that the number of positive brains undergoing decomposition while en route would be balanced by the loss of a proportionate number of negative specimens.

The foregoing tables show that 124 brains received were not examined. The reasons for this are as follows: In 18 cases the brain was destroyed in killing the animal, usually the result of shooting it at close range with a shotgun. In 7 cases the brain was practically destroyed in killing, and partly decomposed. In 98 cases the brain was decomposed to such an extent that examination was impossible. In 1 case the animal was brought to the laboratory alive, observed for two weeks with negative results, and then returned to the owner.

It will be noted that in most instances the reason for nonexamination was decomposition of the brain, and that cases not examined were more numerous in hot weather; hence, the necessity of icing the specimens which are shipped during hot weather is apparent.

It should also be stated that many specimens were in bad condition when received, but one or more tests were made on them and the results of these examinations are embodied in the figures of this report. In general, decomposition or softening of the brain tissues prevents the making of sections; it may cause premature death of inoculated animals and interferes very much with the making of satisfactory smears. On the other hand, it is often possible to obtain smear preparations from the hippocampus major of what seems to be a hopelessly decomposed brain. The hippocampus seems to be one of the last parts of the brain to break down.

Results obtained from the different types of tests.

The foregoing tables show that 124 specimens were not submitted to examination for reasons already stated. Of the 1,003 specimens examined, 153 were submitted to all three tests. The results, as recorded, are shown below in tabular form:

Number of specimens.	Smears.	Sections.	Animal inocula- tion.	Remarks.
5	+ + -	+ - + - +	+ - +	Missed by one microscopic test. Missed by both microscopic tests. Inconsistent finding. Do.
153 (total).				

TABLE V.—Results obtained from the specimens submitted to three tests.

From Table V it is seen that in 139 specimens the findings by all three tests were identical. In 2 specimens the presence of Negri bodies in smears was missed, but detected by the section method and confirmed by animal inoculation. In 8 specimens the presence of Negri bodies was missed by both microscopic methods, but demonstrated by the animal inoculation test. In 3 cases Negri bodies are recorded as present in the smears, though not found on section or inoculation; and in 1 case they are recorded as present in sections, though not demonstrated by smears or inoculation tests. These last four findings are apparently inconsistent, since the animal inoculation test is probably the most reliable of the three. This will be discussed later.

Two hundred and fifty-seven specimens were submitted to two tests with the results shown in Table VI.

Number of specimens,	Smears.	Sections.	Animal inoculations.	Remarks.
10	+	+ + +	+ + + +	Missed by one microscopic test. Missed by both microscopic tests. Inconsistent.

TABLE VI.—Results obtained from the specimens submitted to two tests.

In Table VI there is one inconsistent finding. In 5 cases the smears failed to show Negri bodies, but sections of the same specimens revealed them. In 21 cases the presence of Negri bodies was not detected by the microscope, but animal inoculation proved positive.

The remaining 593 specimens were submitted to but one test. The results are set forth in Table VII.

Number of specimens.	Smears.	Sections.	Animal inoculation.
488	+		
85	_		····- <u>-</u>
3		_	••••••

TABLE VII.—Results obtained from the specimens submitted to one test.

In 488 specimens the presence of Negri bodies was shown in smears and the examination terminated at this point. In 17 cases no attempt was made to make microscopic tests, chiefly on account of the specimens being badly disorganized brains resulting from the killing of the animals by violence. Of these 17 cases, 5 gave positive results by animal inoculation. Three specimens were examined in sections only, on account of the material having been placed in formaldehyde or other fixing reagent before being examined. These all gave negative results. In 85 cases smears were made and examined with negative results, the examination being terminated at this point. Most of these specimens were in varying degrees of decomposition. In some of these cases animal inoculation was attempted, but the concomitant contamination resulted in the death of the the animals within a few days and before rabies could have developed.

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Discussion of results.

Considering the figures given in Tables V and VI we find that of 389 specimens which were submitted to the animal-inoculation test in addition to the microscopic tests, 29 (7.5 per cent) were found positive by inoculation test but negative by the microscope. Also, that of 410 specimens, 36 were found negative on examination by the smear method but later found positive in sections or by inoculation test, making the error of the smear method 8.8 per cent. These examinations were distributed among 11 different men who had charge of the work at various times, the number charged against each man being in proportion to the length of time he was assigned to this work.

The tables also show that in 5 cases (1.3 per cent) a positive finding was reported on microscopic examination which failed of confirmation by animal inoculation, making the total error of the microscopic tests 8.7 per cent. The cases which were reported as positive and which failed to show rabies in the inoculated animals can be attributed in part to an effort to avoid reporting a specimen as "doubtful" or "suspicious." In some of these cases the records show that there was doubt, but that the examiner deemed the microscopic evidence to indicate the presence of Negri bodies. The five specimens thus reported were distributed among four different men and each error was made in the early part of each man's service, showing the conservative tendency of a new man to consider doubtful cases as positive. To illustrate more fully, a copy of the record in each case is quoted.

"No. 127. March 26, 1912. Dog's head from Dr. ——, Staunton, Va. Negri bodies in smears. No Negri bodies in sections. Two guinea pigs innoculated April 8, 1912. Animals all right June 10, 1912. (No. 126 was received at the same time from the same person and this head was too decomposed to permit examination. No mention of the condition of No. 127 is made.)"

"No. 128. April 1, 1912. Dog's head from Dr. —, Staunton, Va. (Same person that sent No. 126 and 127.) Negri bodies in smears. No Negri bodies in sections. Two guinea pigs inoculated April 8, 1912, one animal died April 9, 1912. No symptoms. Other

animal all right on June 27, 1912."

"No. 260. March 31, 1913. Dog's head from Culpeper, Va. Smears negative. A few Negri bodies found in sections. Two guinea pigs inoculated April 2. Both pigs died April 3. Two more pigs inoculated April 3. One pig died September 16, 1913. No Negri bodies found. No symptoms."

25, 1913."

"No. 537. June 1, 1915. Dog's head from —, Shanghai, Va. Suspicious bodies found in smears. Reported positive. Two guinea pigs inoculated June 3, 1915. Both-pigs well and discharged from observation on October, 30, 1915."

Though the animals inoculated from specimens 127 and 128 were observed only for about two and one-half months, they are called negative in this paper since the average incubation period in the 29 cases giving positive result in animal inoculation and negative result on microscopic test was 17.1 days; while in the 44 microscopically positive specimens which were submitted to the animal-inoculation test as a confirmatory procedure the average incubation period was 15.9 days.

The percentage of error as given above has been based upon the assumption that the animal inoculation test gives 100 per cent of positive results. ("Percentage of error" is used with reference solely to the relative accuracy of the three methods and not in the strict mathematical sense.) This, probably, is not always true. To illustrate, let us consider specimen 902, the brain of a cat, which was negative on microscopic test on August 23, 1917. On August 29 two guinea pigs were inoculated from this specimen and on September 22 one pig died. Examination of the pig's brain showed the presence of Negri bodies. The other pig remained well and observation was discontinued on May 18, 1918. Had the first pig died within 5 days of inoculation from extraneous causes the original specimen would have been considered negative. However, such occurrences are negligible and are probably due to error of technique in inoculating one of the animals.

It might be argued that the same proportion of erroneous diagnoses had been made in the 488 specimens reported positive and the 88 reported negative, which were submitted to microscopic test only. While this may be true for the 88 negative cases, many of which were not submitted to animal tests on account of the extremely poor condition of the specimen, it is hardly applicable to the 488 positive specimens. These constitute the easily diagnosed and undoubted cases showing Negri bodies, and if doubt existed they would have been submitted to the animal inoculation test and consequently would have been tabulated as specimens submitted to two or three tests.

The time required for the performance of the different tests is approximately as follows:

Smear test: One hour (frequently less) after arrival of specimen. Section test: Three to four days (a hurried test can be done in a day but will usually be unsatisfactory).

Animal inoculation test: Ten days to six months. After 30 days the probability of a positive result is small.

In conclusion, therefore, it may be stated that, based on 1,003 specimens examined, the correct diagnosis may be obtained on the day of receipt of specimen in 90 per cent of the cases; within five days in about 92 per cent; and at the end of one month practically all will be determined. Hence a negative report on microscopic examination with subsequent inoculation of animals practically becomes a confirmed negative at the end of one month, though it is the rule at present to observe animals for six months.

Summary.

- 1. From February 1, 1909, to April 30, 1919, specimens from 1,127 animals suspected of rabies were submitted to the Hygienic Laboratory for diagnosis.
- 2. One hundred and twenty-four of the specimens received were in such a state of decomposition, or disorganization from violence, that no examination could be made.
- 3. Of the 1,003 specimens examined, 588 showed evidence of rabies, and 415 gave negative results.
- 4. Nearly 82 per cent of specimens submitted and 87.4 per cent of those giving positive results were from dogs.
- 5. The colder months of the year have furnished the greatest number of specimens and the greatest percentage of positive findings.
- 6. Of 389 specimens which were submitted to both microscopic and animal inoculation tests, the microscopic finding was confirmed by the animal test in 91.3 per cent of the cases. The 8.7 per cent error was divided as follows: 1.3 per cent were found positive on microscopic test and negative on inoculation, and 7.4 per cent negative on microscopic test and positive on inoculation.
- 7. Examination by the section method gives slightly more accurate results than by the smear method.
- 8. Of the 406 specimens inoculated into animals there were 79 positive results, the average incubation period being 16.2 days.

VENEREAL DISEASE CONTROL.

PUBLIC HEALTH SERVICE SEEKS COOPERATION OF DENTISTS.

In connection with the campaign for the control of venereal diseases by the various State boards of health acting in cooperation with the Public Health Service, a circular letter, a bulletin, and a pledge card are being sent to each dentist in the United States. He is asked to sign and return the pledge card on which is indicated his willingness to cooperate in this important public health work. The circular letter follows:

2389 October 24, 1919.

To the dentists of the United States:

Veneral diseases are a serious menace to the health of the Nation. The United States Public Health Service, in cooperation with the State boards of health, is making a vigorous campaign for their prevention and control and desires your active cooperation.

The Public Health Service recognizes the especial importance of interesting the dental profession, because syphilis has been transferred to innocent persons through the medium of dental operations.

Furthermore, the public is fast awakening to the fact that the treatment rendered it by the dentist may have a direct bearing on the general health.

The enclosed bulletin outlines your responsibility in this important health work. Will you meet your responsibility and help your State board of health to win this battle against disease?

An agreement card is enclosed. Please sign and mail to-day; no stamp is required.

Respectfully, RUPERT BLUE, Surgeon General.

The following is the text of the bulletin:

An Appeal to Dentists for Cooperation in the Fight Against Venereal Diseases.

The war turned the spotlight on many things heretofore neglected or avoided. No disclosures were more startling than those showing the destructive inroads of venereal disease on the health and efficiency of the Army and Navy.

From the time the United States entered the war in April, 1917, to September, 1918, the loss to the Army from venereal disease represented 2,295,000 days of service.

Now the war is over and the Nation is on its way to a peace basis. Interest begins to turn from the fighting efficiency of the Army to the reconstructive power of industry; and as it turns, this striking fact stands out: All venereal diseases in the Army were caused by conditions in civilian life. The Army and Navy, as organizations, do not tolerate prostitution.

TESTIMONY OF THE ARMY.

Immediately following the declaration of war, the Army Medical Department organized to cope with venercal disease, and one of the first points emphasized by its Surgeon General was that each individual case must be treated under competent medical supervision until cured. He laid special emphasis on two points:

- 1. The ineffectiveness of self-treatment by the use of simple or patent remedies.
- 2. The danger of quack doctors, who advertise to treat so-called private diseases.

Nineteen months of war have shown conclusively the value of proper methods of treating venereal cases in the Army.

MAINTAIN INDUSTRIAL EFFICIENCY.

For the protection of the fighting men as they return home, and to maintain maximum industrial efficiency, venereal disease among the civilian population must be kept under control. There is the same necessity for proper methods of treatment as existed in the service. Self-treatment and quackery must go.

RESPONSIBILITY OF DENTISTS.

Dentists must share the responsibility for preventing the spread of syphilis. This disease is found in all walks of life, all classes of society, and it is too often not recognized when examinations of the mouth are made for dental treatment. Persons who have lesions in the mouth and know they have syphilis, and admit it, do not as a rule seek the services of a dentist unless compelled to do so. Those who have lesions and are unaware of their condition present themselves to the dentist unsuspectingly, and for that reason are a serious menace to the health of the dentist and his clientele.

The ease with which the disease is transmitted is well known to the members of the profession. A break in the skin or mucous membrane, a spirochete gaining entrance and passing into the circulation—and infection occurs. Syphilis is no respecter of persons; neither is it a respecter of tissues. The simple scaling of the teeth may, and usually does, involve a breaking in the continuity of gum tissue and exposes the patient to any infection carried on an unclean instrument. In this connection the Surgeon General, recognizing the extreme requirements of surgical cleanliness and the difficulty with which the germ of syphilis is destroyed, recommends and urges that dentists continue to study and practice the principles of asepsis in all phases of their work. You are reminded that the saliva of those persons infected with syphilis is usually loaded with spirochetes, and that it is not necessary that blood be drawn from a patient in order to infect another through the application of instruments.

Recognizing the various lesions found in the mouth, which are signs of definite diseases, requires clinical knowledge and experience. Many infectious diseases produce lesions in the mouth, and the study of these lesions which might enable the dentist to recognize such infections, as well as the infection of syphilis, is extremely interesting and should be of material benefit to the dentist. It is believed that if the question of venereal diseases were discussed more freely and frequently in study clubs and dental societies, it would be a benefit to society and reflect credit on the dental profession.

Recent investigations have clearly proved that dental structures are an important part of the human economy. The man who treats his case symptomatically is most likely to fail. Dentists have been confronted with increased responsibility in the matter of general health, not merely because they have aspired to greater things but because it has been thrust upon them, and the proposition must be met face to face.

COOPERATION OF PHYSICIANS, DRUGGISTS, AND ADVERTISING MEDIA.

The campaign now being carried on among dentists is similar to those campaigns carried on with physicians, druggists, and advertising media. It should interest you to know that approximately 61,000, or nearly 50 per cent of the physicians of the United States, and that approximately 28,000, or nearly 60 per cent of the druggists of the United States, have signed and returned agreement cards obligating themselves to cooperate; and in addition to these more than 99 per cent of all newspapers and periodicals in the United States carrying advertising have pledged themselves not to carry quack advertising.

WEIGH THESE QUESTIONS.

Will the dental profession accept its share of the burden which this work requires? On receipt of the inclosed franked card, properly signed, the Public Health Service will ask your State board of health to supply you with additional scientific and miscellaneous literature.

RUPERT BLUE,

Surgeon General, United States Public Health Service.

The pledge card contained the following:

Appreciating the seriousness of venereal diseases among the civilian population, as indicated by the reports of the Surgeon General of the Army, I hereby give assurance of my best efforts in cooperating with the United States Public Health Service and my State board of health to reduce the prevalence of venereal diseases, and specifically do I agree:

- 1. To report all venereal disease cases which come under my observation in my practice in accordance with the laws and board of health regulations of my State.
- 2. To advise treatment for all such venereal disease cases which come under my observation, referring them to a clinic or a physician known by me to be competent in the treatment of such cases.

(Name)	
(Street)	• • • • • • • • • • • • • • • • • • • •
(City and State)	
Date	

DEATHS DURING WEEK ENDED OCT. 11, 1919.

From the "Weckly Health Index," Oct. 14, 1919, issued by the Bureau of the Census, Department of Commerce.

Deaths from all causes in certain large cities of the United States during the week ended Oct. 11, 1919, infant mortality (per cent), annual death rate, and comparison with corresponding week of preceding years.

	Population		nded Oct. 1919.	A,	rerage	Per cent under		
City.	July 1, 1918, esti- mated.	Total deaths.	Death rate.1	dea	nual th rate 1,000.2	Week ended Oct. 11, 1919.	ye	evious ar or ears.2
Albany, N. Y.	112, 565	30	13.9	Ç	17.4	10.0	ç	18.9
Atlanta, Ga	201, 732 3 669, 981	48 · 170	12. 4 13. 2	CA	14. 9 15. 6	10. 4 15. 9	CA	16. 1 21. 3
Baltimore, MdBirmingham, Ala	197, 670	40	10.6	A	14.6	27.5	Â	16.0
Boston, Mass	785, 245	171	11.4	Â	15.1	19.3	Â	18.9
Buffalo, N. Y	473, 229	129	14.2	Ā	13. 2	26. 4	Ā	21.8
Cambridge, Mass	111, 432	20	9.4	A	11.1	15.0	A	12. 1
Chicago, I I	2, 596, 681	530	10.6	A	13. 2	18.9	A	21.3
Cincinnati, Ohio	418,022	109	13.6	Č	14.2	11 9.	C	8.0
Cleveland, Ohio	810, 306	164	10.6 11.3	C	10.8 10.2	20. 1 20. 4	·c·	18, 6
Columbus, Ohio	225, 296 130, 655	49 30	12.0	Ă	13.4	30.0	Ă	19.8
Denver, Colo.	130,033	65	12.0	Â	10.7	12.3	A.	15.0
Fall River, Mass	128, 392	30	12.2	Ĉ	13.9	26.7	Ċ	32. 4
Grand Rapids, Mich	135, 459	29	11.2	C	14.5	20.7	c	5. 4
Indianapolis, Ind	290, 389	61	11.0	C	13.8	14.8	Ç	8.0
Jersey City, N. J	318, 770	65	10.6	C	12.5	27.7	g	20.0
Kansas City, MoLos Ange'es, Calif	313, 785 568, 495	81 135	13.5 12.4	C A	13. 6 10. 2	13. 6 9. 6	C A	5.0 12.7
Louisville, Ky.	242, 707	32	6.9	ĉ	15. 2	15. 6	Ĉ	8.6
Lowell, Mass.	109, 081	32	15.3	Ă	18.2	9.4	Ă	30. 1
Memphis, Tenn	154, 759	45	15. 2	Ċ	19.6	11.1	C	5.3
Milwaukee, Wis	453, 481	95	10.9	A	10.5	11.6	A	24.7
Minneapolis, Minn	383, 442	69	9.4	Č	8.5	17.4	ç	11.5
Nashville, Tenn	119, 215	33	14. 4	Ç	11.0	9. 1 20. 9	C	12.0
New Haven, Conn	428, 684 154, 865	91 d 34	11. 1 11. 4	A C	12.0 13.0	17.6	ċ	23. 7
New Orleans, La.	382, 273	97	13. 2	Ä	18.7	14.4	Ă	10.5
New York, N. Y.	5, 215, 879	1,001	10.0	Â	12.9	16. 1	Ã.	16.1
Oakland, Calif	214, 206	55	13. 4	Ã	9.5	5. 5	A	9.8
Omaha, Nebr	180, 264	36	10.4	C	9.9	19.4	C	18.8
Philadelphia, Pa	1,761,371	382	11.3	(t)	14.3	17.0	8	18.0
Pittsburgh, Pa	593, 303	152	13. 4		16.0	11.8 16.7		17. 2
Providence R I	263, 613	54 54	10.7	c	8.6 10.9	13.0	C	15. 7 14. 8
Providence, R. I	160, 719	42	13.6	č	14.1	14.3	č	16.3
Rochester, N. Y	264, 856	60	11.8	č	15.5	13.3	č	22. 1
St. Louis, Mo	779, 951	143	9.6	C	11.7	9.8	Ċ	12.8
St. Paul, Minn	257, 699	37	7.5	Ċ	8.1	10.8	Č	18.0
San Francisco, Calif	478, 530	105	11.4	č	12.1	0.0	ç	6.4
Spokane, Wash	161 404	14		Ç	7.6	7.1	C	8. 7 17. 4
Syracuse, N. Y	161, 404 262, 234	28 48	9. 0 9. 5	C A	15. 1 12. 5	17. 9 10. 4	A.	17. 7
Washington, D. C	401,681	99	12.9	Ã	14.6	11.1	Ã	14. 2
Worcester, Mass	173,650	35	10.5	ĉ	15.6	14.3	ĉ	19.6

Summary of information received by telegraph from industrial insurance companies for week ended Oct. 11, 1919.

Policies in force	41, 484, 574
Number of death claims	6, 635
Death claims per 1,000 policies in force, annual rate.	8. 3

Annual rates per 1,009 estimated population.
 "A" indicates data for the corresponding week of the years 1913 to 1917, inclusive. "C" indicates data for the corresponding week of the year 1917.
 Population estimated as of July 1, 1919.
 Data are based on statistics of 1915, 1916, and 1917.

AUTHORITY TO MAKE HEALTH REGULATIONS IN NEW YORK.

In a case.¹ decided in November, 1916, the New York Supreme Court upheld a regulation issued by the commissioner of public safety of Rochester making it necessary for a milk dealer, before he could obtain a license, to submit to a blood test to ascertain whether he was a carrier of typhoid fever organisms.

The appellate court has recently reversed this decision, holding that under the State laws such a requirement can only be imposed by the legislature or by the city council.²

¹ People ex rel. Schulz v. Hamilton, Comr. of Public Safety, et al., 161 N. Y. Supp. 425; Pub. Health Repts., Jan. 12, 1917, p. 99.

² People ex rel. Schulz v. Hamilton, Comr. of Public Safety, et al., 177 N. Y. Supp. 222.

PREVALENCE OF DISEASE.

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring.

UNITED STATES.

CURRENT STATE SUMMARIES.

Telegraphic Reports for Week Ended Oct. 18, 1919.

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers.

ARKANSAS.		. CONNECTICUT.	
	ses.	Ca	ses.
Diphtheria	5 6	Cerebrospinal meningitis:	
Hookworm	2	Windham County—	
Influenza	19	Woodstock	. 1
Malaria	206	Unicken pox	18
Measles	1	Diphtheria:	
Pellagra	10	Fairfield County—	
Scarlet fever.	35	Bridgeport	. 10
Smallpox	4	Danbury (city)	. 1
Trachoma	1	Danbury (town)	. 1
Tuberculosis	24	Darien	. 3
Typhoid fever	37	* Shelton	. 4
Whooping cough	17	Stamford (city)	6
11 200 ping 00 08 11 11 11 11 11 11 11 11 11 11 11 11 11	•	Stratford	. 5
		Hartford County—	
CALIFORNIA.		Bristol	6
Influenza	35	East Hartford	
Poliomyelitis:		Hartford	
Los Angeles	1	Manchester	
Smallpox:		New Britain	
Alameda	2	Southington	
Crescent	1	Windsor Locks	
Eureka.	3	Litchfield County—	_
Humboldt County	1	Kent	1
Los Angeles County	4	North Canaan	
Los Angeles	4	Middlesex County—	-
Riverside County	6	Chester	1
San Bernardino County	3	New Haven County—	-
San Diego	1	Branford	1
Sonoma County	1	Meriden (eity)	
Ventura County.	1	Meriden (town).	2
Typhoid fever:	-	New Haven	_
Eldorado County	3	Wallingford	
Fresno County	2	Waterbury	
Huntington Fark	1	New London County—	10
Los Angeles	10	New London	3
I os Angeles County	10	Norwich	3
Eanta Monica.	1	Stonington	1
Riverside County	1	Tolland County—	•
Riverside	1	Rockville	3
AM / CI SIGC	1 1	MUCE VILLE	3

connecticut—continued.		CONNECTICUTcontinued.	
Diphtheria—Continued. Windham County—	ases.	Scarlet fever—Continued. New London County—	
		New London County— C	ases
Brooklyn		New London	
Gonorrhea.		Rockville	,
Influenza:	. 20	Syphilis	. 4
Fairfield County—		Tuberculosis	
Newtown	. 1	Typhoid fever:	. 00
Hartford County—		Hartford County—	
Hartford	. 3	Canton	
New Britain		Windsor Locks.	
Litchfield County—	_	Litchfield County—	
. Watertown	. 2	Litchfield	. 1
New Haven County-		Plymouth	
Waterbury	. 1	Watertown	
New London County—		New Haven County-	
New London	. 1	North Haven	. 1
Tolland County—		Whooping cough	. 8
Mansfield	. 1	DELAWARE.	
Windham County—		Chicken pox.	1
Brooklyn	. 1	Diphtheria:	•
Measles:		New Castle	. 9
Fairfield County—		Wilmington	
Bridgeport	. 1	Mumps	
Stamford (city)	. 5	Pneumonia:	_
Hartford County—		Delaware City	. 1
Hartford		Seaford	
New Britain	1	Wilmington	. 1
Litchfield County—		Scarlet fever:	
Plymouth		Georgetown	
Watertown	1	Seaford	
New Haven County—		Townsend	
Hamden	1	Wilmington	6
Milford	2	Georgetown	1
New Haven	9	Tuberculosis:	,
North Haven	3	Lewes.	1
Seymour	2	Wilmington.	3
Waterbury	1	Typhoid fever:	·
Windham County—		Laurel	2
Mocsup—epidemic.		Lewes	1
Plainfield	1	Milford	3
Scarlet fever:	1	Milton	1
Fairfield County—		Smyrna	
Bridgeport	2	Wilmington	1
Darien	1		
Greenwich	2	FLORIDA.	
Shelton	2	Cerebrospinal meningitis	1
Hartford County—	-	Diphtheria	17
Hartford	8	Dysen ery	11
Manchester	1	Influensa	21
New Britain	4	Malaria:	
Rocky Hill	1	Baker County	3
Litchfield County—	1	Bay County	2
Litchfield	1	Calhoun County	2
Plymouth	8	Clay County	2
Middlesex County—		Columbia County	1
Haddam	1	Dade County	1
New Haven County—		De Soto County	3
Hamden	1	Duval County	21
Madison	2	Escambia County	1
Meriden	6	Gadsden County	4
Meriden (town)	2	Jackson County	1
Milford	1	La Fayette County.	6
New Haven	7	Le in County.	5
Wallingford	2	Levy County	9
** (\$15) [1] [1] [1] [1] [1] [1] [1] [1] [1] [1]	TO 1	Anty Country	3

FLORIDA—continued.	**	INDIANA—continued.	
	Cases.		ases.
Madison County		Francisco	
Marion County			. 6
Orange County		Wabash County	
Polk County		Wayne County	. 2
St. Lucie County		Influenza:	
Sumter County		Carroll County	. 1
Taylor County		Dekalb County	
Volusia County		Elkhart County	
Walton County		Fountain County	
Washington County		Franklin County	
Pneumonia	. 24	Greene County	
Typhoid fever	. 13	Hancock County	3
anonari		Henry County	4
GEORGIA.		Jasper County	
Cerebrospinal meningitis	. 1	Marion County	2
Chicken pox		Martin County	5
Conjunctivitis (acute infectious)		Sullivan County	
Dengue		Gonorrhea	75
Diphtheria		Measles:	
Dysentery (bacillary)		Al'en County	1
Gonorrhea.		Dekalb County	1
Hookworm		Henry County	1 1
Influenza.	-	Marion County	1
Malaria		St. Joseph County	1
Measles	. 6	Tippecanoe County	1
Measles (German)	. 1	Vigo County	ī
Mumps	. 13	Scarlet fever:	_
Paratyphoid fever		Adams County	1
Pneumonia (acute lobar)		Blackford County	3
Scarlet fever		Clinton County	2
Septic sore throat		Dearborn County	2
SmallpoxSyphilis		Decatur County	15
Tuberculosis (pulmonary)		Delaware County	3
Typhoid fever		Elkhart County	2
Whooping cough		Floyd County Franklin County	1 2
	-	Fulton County	1
INDIANA.		Grant County	2
Cerebrospinal meningitis:		Greene County	ī
Fulton County	1	Hancock County	1
Diphtheria:	- 1	Henry County	1
Allen County	5	Howard County	1
Blackford County	4	Huntington County	1
Daviess County	2	Jasper County	3
Dekalb County	3	Knox County	2
Elkhart County	7	Lake CountyLawrence County	1 1
Henry County	i		21
Howard County	il	Noble County	1
Jackson County	î l	Orange County	4
Lake County	i	Owen County	1
Lawrence County	1	Porter County	4
Madison County	1	Randolph County	1
Marion County	19	Ripley County	5
Marshall County.	1	Rush County	1
Montgomery County	2	Shelby County	8
Owen County	3	Steuben County	2
Parke County	1	Sullivan County	1
Posey County	2 1	Tippecanoe County Vigo County	7
Rush County	1	Wabash County	6 5
St. Joseph County	il	Wayne County	1
	- '		-

INDIANA—continued.		Iowa—continued.	
Smallpox:	Cases	. Scarlet fever:	ases
Dearborn County		Boone.	. 4
Fountain County		Boone County.	3
Fulton County		Burlington	9
Grant County		Council Bluffs	. 6
Huntington County		Dallas County	1
Jackson County		Davenport	. 1
Jasper County		Des Moines.	. 10
Jefferson County	1	Emmetsburg	. 2
Porter County	1	Harrison County	. 3
St. Joseph County	2	Jasper County.	. 1
Steuben County	2	Maxwell	. 1
Tippecanoe County	4	Ottosen	. 2
Tipton County	1		. 1
Vermilion County	4	Wayne County	. 6
Warren County	1	Smallpox:	
Syphilis	72		. 2
Typhoid fever:		Clinton County	. 1
Allen County	3	Davenport	9
Decatur County	1		. 1
Franklin County		Harrison County.	. 1
Grant County		Lost Nation	. 3
Hancock County		Syphilis	ຸ່ວ
Howard County	2	, p	90
Huntington County		KANSAS.	
Lawrence County			
Marion County	1	Cerebrospinal meningitis:	
Martin County		Eureka	1
		Doniphan	1
Orange County		Long Island	1
St. Joseph County		Diphtheria	97
Steuben County		Influenza	34
Sullivan County		Scarlet fever	65
Tippecanoe County		Smallpox	6
Vigo County			٠
Wabash County	1	LOUISIANA.	
IOWA.			
Chancroid:		Cerebrospinal meningitis	
Grinnell	. 1	Chancroid	25
Chicken pox		Diphtheria	16
Diphtheria:		Gonorrhea.	153
Benton County	. 3	Influenza.	37
Cedar Rapids	. 3	Poliomyelitis	1
Council Bluffs	. 4	Smallpox	7
Davenport	. 2	Syphilis	112
Des Moines	. 10	Typhoid fever	16
Dubuque	. 3		
Fort Dodge	. 5	MAINE.	
Garner	. 1	Carabragainal maningities	
Hancock County	. 1	Cerebrospinal meningitis:	_
Iowa Falls.		South Berwick	1
Jasper County.	. 1	Chancroid	1
Mason City.	. 1	Chicken pox.	3
Norway	. 2	Diphtheria:	
Norway		Brunswick.	1
Panora	. 1	Fort Fairfield	1
Red Oak	. 3	Lewiston	5
Story County	. 2	Monmouth	1
Watkins			64
Gonorrhea.	. 75	Influenza:	
Influenza:		Bath	1
Maxwell	. 1	Corinna	3
Story County	. 1	Portland	4
Measles:		South Berwick	2
Boone County.	. 4	Measles:	
Poliomyelitis:		Portland	1
Wapello County	. 1	Mumps	2

MAINE—continued.		NEW YORK.	
Scarlet fever:	ases.	(Exclusive of New York City.)	
Benton.		Cerebrospinal meningitis:	ases
Bingham		Schenectady.	
Brunswick	_	Diphtheria:	
Concord		Erie County	. 12
Kittery		Scattering	
Portland		Gonorrhea	
Sherman		Influenza	. 3
Smallpox:	2	Measles	. 65
Auburn		Pneumonia	. 39
Portland		Scarlet fever	. 135
Pittsfield		Smallpox:	
Syphilis Trachoma:		North Collins	. 2
Brunswick	1	Syphilis	. 116
Tuberculosis.		Typhoid fever	
Typhoid fever:	12	Whooping cough	147
Bath	1	NORTH CAROLINA.	
Livermore			_
Portland		Cerebrospinal meningitis	
Rockland		Chancroid	
Saco.		Chicken pox	
Warrville.		Diphtheria	
Whooping cough		Dysentery (bacillary)	
	•	Gonorrhea.	
MASSACHUSETTS.		Measles	
Anthrax	1	Measles (German)	
Cerebrospinal meningitis	4	Ophthalmin neonatorum	
Chicken pox	65	Paratyphoid	
Conjunctivitis (suppurative)	4	Pneumonia (lobar)	
Diphtheria	221	Poliomye'itis.	
Dysentery		Scarlet fever.	
Gonorrhea	121	Septic sore throat	29
Influenza		Smallpox	
Measles	131	Syphilis	
Measles (German)	8	Trachoma.	
Mumps		Typhoid fever.	
Ophthalmia neonatorum		Whooping cough	
Pneumonia			
Poliomyelitis (anterior)	4	OHIO. Diphtheria:	
Scarlet fever		Cincinnati	99
Septic sore throat	4	I ima.	
Smallpox:		Typhoid fever:	10
Boston	1	Piekaway County, Scioto Township;	
Syphilis		Institution for Feeble-Minded	4
Trachoma	1	,	-
· · · · · · · · · · · · · · · · · · ·	3	VIRGINIA.	
Tuberculosis (pulmonary) Tuberculosis (other forms)		Smallpox: Grayson County	3
Typhoid fever	39	Greene County	
Whooping cough		King George County.	
	•	Rockingham County, several.	•
MINNESOTA.		WASHINGTON.	
Poliomyelitis	1	Chicken pox	07
Smallpox (new foci):		Diphtheria	27
Big Stone County, Prior Township	1	Gonorrhea.	44
Freeborn County, Myrtle	1	Influenza.	34 6
MONTANA.		Mcasles.	
		Mumps	8 75
Diphtheria	8	Pneumonia	3
Scarlet fever	35	Scarlet fever.	45
Smallpox	4	Smallpox.	77
Typhoid fever	10	Syphilis	8
NEW JERSEY.		Tuberculosis.	
Influenza	42	Typhoid fever	5
Pneumonia	39	Whooping cough	

WEST VIRGINIA.		WEST VIRGINIA—continued.	
Diphtheria: Ca	ses.	Smallpox—Continued.	ases.
Bluefield	5	Wheeling	
Buckhannon	2	Williamstown	. 1
Charleston	4	Typhoid fever:	
Charles Town.	1	Hinton	. 1
Clarksburg	2	Huntington	. i
Elkins.	1	Ripley	. 3
Fairmont	9	Wheeling.	. 3
Grafton	3		
Hinten	3		
Huntington	14	Wisconsin.	
Keyser	1	Chancroid	. 3
Martinsburg.	4	Cerebrospinal meningitis:	
Montgomery	1	Milwaukee	. 1
Morgantown	2	Chicken pox:	
Parkersburg	5	Milwaukee	. 9
Parsons	3	Scattering	. 7
Weston	3	Diphtheria:	
Wheeling.	1	Milwaukee	. 33
Williamson.	5	Scattering	
Influenza:	_	Erysipelas:	
Charleston	1	Milwaukee	. 1
Keyser	3	Influenza.	• -
Moorefield.	2	Gonorrhea	
Morgantown.	5	Measles:	. 200
Measles:	-	Milwaukee	. 11
Wheeling.	1	Scattering	•
Scarlet fever:	_	Poliomyelitis:	
Bluefield	3	Milwaukee	. 1
Charleston	2	Scattering.	_
Clarksburg	3	Searlet fever:	, ,
Fairmont	5	Milwaukee	. 29
Grafton.	1	Scattering	
Huntington	ī	Smallpox:	-
Martinsburg.	ī	Milwaukee	. 6
Montgomery.	ī	Scattering	
Morgantown.	3	Syphilis	
Smallpox:	٠	Tuberculosis:	
Clarksburg	2	Milwaukee	. 15
Grafton	3	Scattering	
Huntington	15	Typhoid fever:	
Montgomery	1	Milwaukee	. 1
Parkersburg.	1	Scartering	
Piedmont	1	Whooping cough	
* ************************************	-	11 TOOLTHE CORETTION	, 00

SUMMARY OF CASES REPORTED MONTHLY BY STATES.

Tables showing by counties the reported cases of cerebrospinal meningitis, malaria, pellagra, pollomyelitis, smallpox, and typhoid fever are published under the names of these diseases. (See names of these and other diseases in the table of contents.)

The following monthly State reports include only those which were received during the current week. These reports appear each week as received.

- State.	Cer- ebro- spinal menin- gitis.	Diph- theria.	Mala- ria.	Measles.	Pel- lagra.	Pol- iomy- elitis.	Scarlet fever.	Small- pox.	Ty- phoid fever.
1919. Colorado (August) Maryland (September) Nebraska (September) West Virginia (September) Wisconsin (September)	1 2 1 2 3	38 215 63 270 210	22	9 24 2 8 60	4	2 27 7 8 37	12 138 32 196 213	75 7 57 50 82	33 344 11 271 21

ACTINOMYCOSIS.

Nebraska Report for September, 1919.

During September, 1919, one case of actinomycosis was reported in Nebraska.

CEREBROSPINAL MENINGITIS.

State Reports for August and September, 1919.

Place.	New cases reported.	Place.	Newcases reported.
Colorado (August): El Pase County Maryland (September): Anne Arundel County Baltimore County— Catonsville. Total. Nebraska (September): Saline County.	1 1 2 2 1	West Virginia (September): Cabell County Monongalia County Total Wisconsin (September): Milwaukee	1 1 2 3

City Reports for Week Ended Oct. 4, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Baltimore, Md. Birmingham, Ala Boston, Mass Charlotte, N. C. Chicago, Ill. Cleveland, Ohio. Fort Wayne, Ind. Grand Rapids, Mich	4 , 1	2	Kansas City, Mo Lowell, Mass. Lynn, Mass. Montgomery, Ala New York, N. Y. Philadelphia, Pa St. Louis, Mo.	1	1 5 1

DIPHTHERIA.

See Telegraphic weekly reports from States, p. 2394; Monthly summaries by States, p. 2399; and Weekly reports from cities, p. 2409.

INFLUENZA.

Cases Reported by State Health Officers, Week Ended Oct. 18, 1919.

Arkansas	19	Louisiana.	37
California	35	Maine	10
Connecticut	11	Massachusetts	43
Florida	21	New Jersey	42
		New York	
Indiana	32	Washington	6
Iowa	2	West Virginia	11
Kansas	34	Wisconsin	8

LEPROSY.

California and Colorado.

During the month of August, 1919, one case of leprosy was reported in Otero County, Colo., and during the week ended October 4, 1919, one case was reported at San Francisco, Calif.

LETHARGIC ENCEPHALITIS.

Chicago, Ill., Week Ended Oct. 11, 1919.

During the week ended October 11, 1919, one case of lethargic encephalitis was reported in Chicago, Ill.

MALARIA. Maryland Report for September, 1919.

Place.	New cases reported.	Place.	New cases reported.
Maryland: Anne Arundel County— Mayo. Calvert County— Adelina. Lower Marlboro. Charles County— White Plains. La Plata. Dorehester County— Hills Pennt. Eldorado. Kent County— Tolehester, R. D. Rock Hall. Prince Georges County— Townshend. Congress Heights.	1 1 1 1 1 1 1 1	Maryland—Continued. St. Marys County— Beauvus. Hollywood. Talbot County— Witman, R. D. Wicomico County— Clara. Bivalve. Salsbury. Mardella. Worcester County— Pocomoke City, R. D. Total.	2 2 1 1 1 2 1 1 22 2

City Reports for Week Ended Oct. 4, 1919.

· Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Anniston, Ala. Beaumont, Tex Birmingham, Ala. Boston, Mass. Cleveland, Ohio Dallas, Tex Danville, Va Independence, Mo Little Rock, Ark Los Angeles, Calif Memphis, Tenn	1 2 3 1 20 10 1	2	Pine Bluff, Ark	1 10 10 2 9 1	1

MEASLES.

See Telegraphic weekly reports from States, p. 2394; Monthly summaries by States, p. 2399; and Weekly reports from cities, p. 2409.

PELLAGRA.

Maryland Report for September, 1919.

During September, 1919, pellagra was reported in Maryland as follows: Baltimore, 2 cases; Hyattsville, Prince Georges County, 1 case; Crisfield, Somerset County, 1 case.

City Reports for Week Ended Oct. 4, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Birmingham, Ala Bluefleld, W. Va Brunswick, Ga Charleston, S. C Mobile, Ala		1	Montgomery, Ala	1	

PLAGUE-INFECTED GROUND SQUIRRELS.

Alameda and Contra Costa Counties, Calif.

One plague-infected ground squirrel was reported found in Alameda County on September 4, and another on September 22, 1919. On September 17, 1919, one was reported found in Contra Costa County, Calif.

PNEUMONIA.

City Reports for Week Ended Oct. 4, 1919.

	Lol	ber.	All	forms.		Loi	bar.	Alla	orms.
Place.	Cases.	Deaths.	Cases.	Deaths.	Place.	Cases.	Deaths.	Cases.	Deaths.
Anderson, Ind. Atlanta, Ga. Baltimore, Md. Baltimore, Md. Belleville, N. J. Birmingham, Ala Boston, Mass. Brookline, Mass. Brookline, Mass. Buffalo, N. Y. Burlington, Iowa Burlington, Iowa Burlington, Vt. Cambridge, Mass. Camden, N. J. Canton, Ohio Charleston, S. C. Chelsea, Mass. Chicago, Ill Cincinnati, Ohio. Cleveland, Chio.	9 1 20 1 1 2 1	1 1 6 1 4 1 1 1 5 12		2	Lincoln, Nebr. Little Rock, Ark. Los Angeles, Calif. Louisville, Ky. Lowell, Mass. Lynn, Mass. Memphis. Tenn Methuen, Mass. Midletown, N. Y. Milwaukee, Wis. Minreapolis, Minn Newark, N. J. New Bedford, Mass. Newhuryport, Mass. New Haven, Conn New Orleans, La. New York, N. Y. North Attleboro, Mass. Norwalk, Conn Oakland, Calif. Oaklahoma City, Okla.	1 1 3	3 3 1 2	11 1 15 2	1 1 2 1
Council Bluffs, Iowa Covington, Ky Covington, Ky Counberland, Md. Dayton, Ohio Denver, Colo. Detroit, Mich East Chicago, Ind El Paso, Tex Fort Worth, Tex Fort Worth, Tex Framingham, Mass Fremont, Ohio Fresno, Calif Grand Bapids, Mich Green Bay, Wis Haverhill, Mass Jamestown, N. Y Jersey City, N. J Kansas City, Kans Kansas City, Kans Kansas City, Kokomo, Ind Lackawanna, N. Y Lawrence, Mass Lexington, Ky	1 1 9 1 2 3 1 1	1 2 1 2 1 1	3	1 2 14 	Omaha, Nebr Passaic, N. J. Paterson, N. J. Paterson, N. J. Pawtucket, R. I. Peoria, Ill. Philadelphia, Pa Pittsfield, Mass Quircy, Mass. Racine, Wis Raleigh, N. C. Riverside, Calif. Rochester, N. Y. Ralme, N. Y. Salem, Mass. Salt Lake City, Utah San Bernardino, Calif. San Francisco, Calif. San Francisco, Calif. Savannah, Ca. Somervilk, Mass. Springfield, Mass Springfield, Mass Springfield, Mass Syracuse, N. Y. Tauntor, Mas. Wilmington, N. C. Worcester, Mass.	35 1 1 1 1 11 11	1 2 19 1 1 1 1 1 2 2	1	1 1 3

POLIOMYELITIS (INFANTILE PARALYSIS).

State Reports for August and September, 1919.

Place.	New cases reported.	Place.	New cases re- ported.
Colorado (August): Clear Creek County	2	West Virginia (September)—Continued. Mercer County. Wyoming County.	
Maryland (September): Baltimore	20	Total	
CumberlandBaltimore County—	1	Wisconsin (September): Barron County	
Sparrows Point	1	Dane County Fond du Lac County Grant County	
Queen Annes County— Ingleside, R. D	1	Green Lake County	
Total	27	Juneau County Kenosha County	
Vebraska (September): Brown County Colfax County	2	Lafavette County	
Cuming County Douglas County	1	Ozaukee County	
Kearney County	1	Rock County	
Total	7 	Waukesha County	
West Virginia (September): Berkeley County	1 1	Total	3

City Reports for Week Ended Oct. 4, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Baltimore, Md Birmingham, Ala Chicago, Ill Columbus, Ohio Kansas City, Mo Lowell, Mass Methuen, Mass	2 1 1 1	3 1	New York, N. Y,	1 1 1	1 1 2

RABIES IN ANIMALS.

Dayton, Ohio, and West Orange, N. J.

During the week ended October 4, 1919, one case of rabies in animals was reported at Dayton, Ohio, and one case was reported at West Orange, N. J

RABIES IN MAN.

New York, N. Y., and Salt Lake City, Utah.

During the week ended October 4, 1919, one case of rabies in man was reported at New York, N. Y. On October 9, 1919, one death from rabies was reported at Salt Lake City, Utah.

SCARLET FEVER.

See Telegraphic weekly reports from States, p. 2394; Monthly summaries by States, p. 2399; and Weekly reports from cities, p. 2409.

SMALLPOK. State Reports for August and September, 1919—Vaccination Histories.

			,	Vaccination l	nistory of cas	es.
Place.	New cases reported.	Deaths.	Number vaccinated within seven year preceding attack.	DEVOCE INCL.	Number never suc- cessfully vaccinated	Vaccination history no obtained on uncertain.
Colorado (August):						
Alamosa County	1	 	.	.	. 1	ļ
Arapahoe County	1			·		
Boulder County	1				. 1]
Cheyenne County	2	• • • • • • • • • •			.] 2	ļ
Crowley County	5		•		1 1	
Delta County	35	•••••			26	1
Denver El Paso County	า๊า			-	1	
Grand County	5		i		1 1	
Gunnison County	ă l		l i		3	1
Huerfano County	4	•••••	1		3	
Jefferson County	2				2	
Larimer County	6				1 6	
Las Animas County	1				1	
Summit County	1	• • • • • • • • • •			1 1	
Weld County	2				2	
Total	75		14	2	56	3
Maryland (September): Dorchester County— Cambridge	4				4	
Garrett County	2				2	
BittingerQueen Annes County— Centerville, R. D	1				1	
Total	7				7	
Wisconsin (September):						
Adams County	3 5	• • • • • • • • • • • • • • • • • • • •	i	2	3 2	
Barron County	i l			ī		
Calumet County	3 .					8
Chippewa County	1).					1
Clark County	5 .		3	2		
Dane County	2	• • • • • • • • • • •	2	• • • • • • • • • • • • • • • • • • • •		
Fond du Lac County	7	• • • • • • • • • • • •		••••••	1 2	•••••
Forest County	11	• • • • • • • • • • • • • • • • • • • •		•••••	1	•
Green County	i				•	1
Jefferson County	î					i
Kenosha County	1			1		
Manitowoc County	2 5				2	
Marathon County	5 .				5	· · · · · · · · · · · · · · · ·
Milwaukee County	5 .					
Monroe County	6 .					•
Oconto County	3 9	•••••••				3
Portage County	9 .	•••••	6	••••••	4]	5
Racine County	6 .		6			••••••••
Sheboygan	4	•••••	• • • • • • • • • • • • • • • • • • • •			
Trempealeau County	i i	••••••	•••••	····i	*	• • • • • • • • • • • • • • • • • • • •
Winnebago County	3 .					3
Wood County	5 .			2	3	
Total	82		12	9	27	34

SMALLPOX—Continued.

State Reports for September, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Nebraska: Cass County Custer County Dodge County Dougles County Lanaster County Saunders County Richardson County West Virginia: Barbour County Braxton County Fayette County Harrison County	4 1 15		West Virginia—Continued: Lewis County McDowell County Marion County Mercer County Mineral County Mingo County Monongalia County Poeshontas County Putnam County Raleigh County Taylor County Wood County Wyoming County	2 5 1 1 1 8 1 2 1 2 2 6 4	

City Reports for Week Ended Oct. 4, 1919.

. Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Atchison, Kans. Baton Rouge, La. Bellin; ham, Wash. Boise, Idaho. Chicago, Ill. Cincinnati, Ohio. Cleveland, Ohio. Dallas, Tex. Davenport, Iowa Detroit, Mich. Eureka, Calif. Everett, Wash. Fairmont, W. Va. Findlay, Ohio. Fond du Lac, Wis. Green Bay, Wis. Grand Rapids, Mich. Indianapolis, Ind. Kokomo, Ind. Lincohn, Nebr. Los Angeles, Calif.	1 1 4 1 1 3 2 1 1 1 1 1 1 3 2 2 3 2 3 2 3 3 2 3 3 3 3		Rishmond, Va. Rocky Mount, N. C. Sacramento, Calif. St. Joseph, Mo. St. Paul, Minn. Salt Lake City, Utah San Diego, Calif. San Francisco, Calif. Seattle, Wash. Sioux City, Iowa. Spokane, Wash. Stockton, Calif. Waco, Tex.	1 1 2 9 2 1 1 10 2 1 8 7	

TETANUS.

City Reports for Week Ended Oct. 4, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Baltimore, Md Baton Rouge, La Boston, Mass Mobile. Ala Muscatine, Iowa Nashua, N. H	1 1	3	New Haven, Conn New Orleans, La. New York, N. Y. Portsmouth, Va. Rome, Ga. Springfield, Mo.	1	1 1 1

TUBERCUI.OSIS.

See Telegraphic weekly reports from States, p. 2394; and Weekly reports from cities, p. 2409.

TYPHOID FEVER.

State Reports for August and September, 1919.

Place.	New cases reported.	Place.	New c
lorado (August):		Maryland (September)—Continued.	
Cheffee County	. 1	Cecil County—	l
Denver County	. 14	Calvert	.[
El Paso County. Huerfano County.		Charles County-	•
Huerfano County	2 1	Tronside	ł .
Kit Carson County		Hughesville. Allens Fresh	
Larimer County	. 2	Allens Fresh	1
Las Animas County	.! 4.1	Dorchester County	ī
Pueble County	. 1	#ederalshipp. K. D	1
Summit County	. 1	Cambridge. East New Market	4
Weld County	. 2	East New Market	
		Finchville. Galestown, R. D.	1
Total	.[33	Galestown, R. D	1
		Frederick County—	1
	1 1	Frederick	1
ryland (September):	1 1	Thurmont	l
Baltimore	73	Brunswick	1
Allegany County	1 .01	Walkersville Hyattstown, R. D	I
Allegany County— Cumberland Allegany Hospital	9	Hvottstown R D	I
Allogony Uconitel	3 1	Hermony	i
Western Maryland Hospital	i	Ademetern P D	1
Tonocoming	1 4	Point of Rocks, R. D.	'
Vount Samma	1	Mount Pleasant R D	ł
Manlerida	l il	Myersville	1
Lonaconing Mount Savage Mapleside Midland	i	Hyattstown, R. D. Harmony. Adamstown, R. D. Point of Rocks, R. D. Mount Pleasant, R. D. Myersville. Knoxville, R. D. Emmitsburg, R. D. Middletown, R. D. Montevue Hospital. Mountain Dale, R. D. Sabillasville, R. D. Walkersville, R. D. Burkittsville, R. D. Burkittsville, R. D. Bethel	l
Luke Hill	1 1	Emmitshare R D	l
Panhlin	i	Widdletown R D	Ī
Franklin Keyser, R. D Westernport National	l il	Montovuo Poerital	ł
Wastern and	il	Mountain Dala D D	
w esternport	1 1	Cabillaguille D D	ł
National	1	Well-and the D. D.	l
Gilpen	1	Walkersville, R. D.	
Anne Arundel County—		Burkittsvine, R. D	ł
Odenton, B. D	5	Myersville, R. D	l
Odenton, R. D. Brooklyn, R. D. Downs.	1	Bethel	ŀ
Downs	1	Garrett County—	ł
Harwood	1 1 1	Kitzmiller	}
Sudley, R. D	1	Kitzmiller Keyser, R. D	
Baltimore County—	1 1	Harford County	l
Sudley, R. D. Baltimore County— Catonsville.	. 5	Havre de Grace. Havre de Grace, R. D.	
Towers	1 11	Havre de Grace, R. D	ł
Phoenix Overlea, R. D Weisburg, R. D	1	Joppa, R. D. Stafford, R. D. Kalmia	
Overlea, R. D	1	Stafford, R. D.	
Weisburg, R. D.	1	Kalmia	
Sparks Timonium Woodlawn, R. D. Garrison Lutherville.	1	Waterville	
Timonium	1	Howard County—	i
Woodlawn, R. D	1	Woodstock	i
Garrison	1	Elk Ridge, R. D	1
Latherville	2	Cooksville, R. D. Woodbine, R. D.	
Reisterstown	ī	Woodbine, R. D	
Summerfield	ī	Kent County—	
Sparrows Point		Rock Hall	
Colvert County-	- H		
Calvert County— Sunderland	1	Sassafras	
Holland Point	2	Montgomery County—	
Dest Describic	1 1 ti	Rockville, R. D.	
Wilson Chesapeake Beach Poplars. Phum Point.	ī	Chestertown, R. D. Sassafras. Montgomery County— Rockville, R. D. Cedar Grove, R. D. Gaithersburg, R. D. Silver Spring. Prince Georges County— Berwyn Heights. Seat Pleasant Accokeek, R. D. Queen Annes County—	
Chesaneaka Beach	î	Gaithersburg, R. D.	
Ponlare	6	Silver Spring	
Phim Point	ĭ	Prince Georges County—	
		Berwyn Heights	
Danton -	2	Seat Pleasant	
Denton, R. D.	î l	Accokeek, R. D.	
Goldsboro	i	Queen Annes County—	
Vadara ichura	11		
Federalsburg. Federalsburg, R. D. Two Johns, R. D.	**	Centreville, R. D. Millington, R. D. Burrisville, R. D.	
Maria Johns D D	2 1	Millington, R D	
		Burrisville, R. D.	•
Bethlehem	3	Chestertown, R. D.	•
rangely, R. D.	2	Andersons Corner, R. D.	
Hynson, K. D	1	Templeville, R. D.	
Burrsville, R. D	1	St Marye County	
Ridgely, R. D. Hynson, R. D. Burrsville, R. D. American Corner, R. D. Mount Zion, R. D. Preston, R. D. Carroll County.	1	St. Marys County— Cedar Point Somerset County— Kingston	
Mount Zion, R. D	1	Company County	
Preston, R. D	1	Somerset County—	
		Kingston	
Union Bridge Eldersburg, R. D.	5	Marion	
Eldersburg, R. D	5	Taipot County—	
Okianoma	1	Talbot County— Easton, R. D. Trappe, R. D. Windy Hill.	
Westminster	1	Trappe, R. D	

TYPHOID FEVER—Continued.

State Reports for August and September, 1919-Continued.

Place.	New cases reported.	Place.	New case reported.
Maryland (September)—Continued.		West Virginia (September)—Continued	
Washington County-		Hampshire County	
Hagerstown	38 3 2 1	Hancock County	1
Hagerstown, R. D.	3	Hardy County	1 3
Hagerstown, R. D. Washington County Hospital	3	Harrison County	22 23 5 6
Boonsboro	1	Jackson County	- 2
Hancock	1	Jefferson County	8
Williamsport Sharpsburg, R. D. Keedysville, R. D.	1 3 1 2	Kanawha County	23
Sharpsburg, R. D] 3	Lewis County	
Keedysville, R. D	1	Logan County	1 8
Maugansville	2	McDowell County	12
Mangansville	1	Marion County	8
Wilson, R. D	1	Marshall County	5
Wilson, R. D. Cavetown, R. D.	1	Mason County	1
Wicomico County	1	Mercer County	} • 3
Salisbury	7	Mineral County	1 8
Salisbury. Salisbury, R. D. Hebron.	1	Mingo County	1 2
Hebron	. 2	Monongalia County	2
Worcester County-		Monroe County.	1 2
Box Iron	2	Morgan County	i i
Eden, R. D	1	Ohio County	
Berlin		Pendleton County.	ì
Newark R D	1 1	Pocahontas County	1 1
Snow Hill, R. D.	1	Preston County.	
Ocean City R D	Ī	Putnam County	
Snow Hill, R. D. Ocean City, R. D. Girdletree, R. D.	Ī	Raleigh County	
St. Martins.	Ī	Roane County	
Bishopville		Summers County	
Distrop vinc			11
Total	344	Tyler County	
±0141	011	Upshur County	3
Nebraska (September):		Webster County	1 9 4 2 8
Burt County	1	Wetzel County	4
Cuming County		Wood County.	9
Douglas County	5	Wyoming County	•
Lancaster County	5	m	271
Morrill County	5	Total	2/1
Polls County	2 2 2 1		
Polk County	2	Wisconsin (September):	_
Daniel County		Barron County	1
Total	11	Columbia County	1
TOTAL		Dane County	1
West Winding (Sontombor):		Kenosha County	2
West Virginia (September): Barbour County	2	Marathon County	1
Boone County	6	Milwaukee County	4
Doore County		Ozaukee County	1
Braxton County	11	Polk County	1
Calhoun County	1	Rock County	1 1 2 1 4 1 1 1 1
Clay County	1	Rusk County	a
Dedd-idea County	2 2	Walworth County	
Doddridge County	าเ	Washington County	4
Fayette CountyGilmer County	15		
Gumer County	19	Total	21
Greenbrier County	4		

City Reports for Week Ended Oct. 4, 1919.

Place.	Cases.	Deaths.	Place.	Cases.	Deaths.
Aberdeen, S. Dak Akron, Ohio. Albany, N. Y Ann Arbor, Mich Atlanta, Ga Baltimore, Md Baton Rouge, La Bedford, Ind Birmingham, Ala Bloomfield, N. J Bluefield, W. Va Boston, Mass Brazil, Ind Brookline, Mass Buffalo, N. Y	3 11 3 3 11 1 1 4 1 1 6	1 1	Cairo, III Canton, Ohio Cape Girardeau, Mo Cedar Rapids, Iowa Charlotte, N. C Chicago, III Cincinnati, Ohio Cleveland, Ohio Coffeyville, Kans Columbia, S. C Columbus, Ga Concord, N. H Dallas, Tex Danville, III Danville, Va	1 1 1 11 2 2 1 1 2 1	1

TYPHOID FEVER—Continued. City Reports for Week Ended Oct. 4, 1919—Continued.

,	Cases.	Deaths.	Place.	Cases.	Deaths
Dedham, Mass	1		Parkersburg, W. Va	1	
Denver, Colo	. 2	i	Paterson, N. J	1	
Detroit. Mich	. 5	2			
Detroit, Mich Duluth, Minn	2	Ž	Philadelphia, Pa	18	İ
Ilmina NI V	1 1	1	Portland, Me	-3	
l Poso Toy	1 -	·····i	Portland, Oreg		ļ
Coirmont W Vo	1	ļ	Portemouth Ohio	3	Į.
El Paso, Tex. Sairmont, W. Va. Sall River, Mass.	1 1		Portsmouth, Ohio	3	
lint, Mich	1 2		Providence, R. I	, 3	
ort Wayne, Ind	3		Pueblo, Colo.	4	
			Rahway, N. J.		
reeport, Illresno. Calif.		1	Dana Non	. 2	
resno, Calif	1 1	1	Reno, Nev	1	
alesburg, Ill	1 !		Richmond, Va		
alveston, Tex	1 1		Roanoke, Va	1	
rand Rapids. Mich		1	Rochester, N. Y	4	
reat Falls, Mont	2		Rockford, Ill	1	
reen Bay. Wis	1		Rome, Ga	1	
laverhill, M iss			Kome, N. Y		
ndianapolis, Ind	1		Sacramento. Calif	2 3 8	
ersey City, N. J	3		St. Joseph, Mo	3	
alamazoo, Mich	1 1		St. Louis, Mo	8	
ansas City, Mo	3	2	St. Paul, Minn	3 (
noxville, Tenn	1 1	1	Salt Lake City, Utah	41	
ima, Ohio	3	_	San Francisco, Calif	il	
eng Branch, N. J	2 2		Seattle, Wash	i	
os Angeles, Calif	2	1	South Bend, Ind	ī	
ouisville, Ky	5 [ī	Southbridge, Mass.	3	••••••
vnchburg, Va	ĭl		Spokane, Wash	3 2 2 2 2	•
ılden, Mass	i i		Springfield, Mass	5 !	
arquette, Mich	1		Springfield, Ohio	5	
emphis, Tenn	5		Syracuse, N. Y.	3	
ilwaukee, Wis	2		Tounton Moce	ĭ	
obile, Ala	ııı		Taunton, Mass	, i	••••••
organtown, W. Va			Topeka, Kans	î	
ount Vernon, N. Y			Trong N V	2	
ount vernon, N. 1		,	Troy, N. Y.	3	
ashville, Tenn			Tuscaloosa, Ala	3	
ewark, N. J			Vancouver, Wash	1	• • • • • • • • • • • • • • • • • • •
ew Britain, Conn			Walla Walla, Wash		• • • • • • • • • • •
ewburgh, N. Y	1)		Waltham, Mass	3	••••••
ew Orleans, La	4	1	Washington, D. C	9	
AW YORK N Y	44	5	Watertown, Mass	1	
iagara Falls, N. Y	1		Westfield, Mass		
orfolk, Va	4 1		White Plains, N. Y		• • • • • • • • • •
orwalk, Conn		1	Wichita, Kans	2	
akland, Califklahoma City, Okla	1		Wilmington, Del	1	
klahoma City, Okla	2		Wilmington, N. C	2	
maha, Nebr	2	2	Winston-Salem, N. C	īl	
range, N. J.		ĩ	Yakima, Wash	2	

TYPHUS FEVER.

Nebraska Report for September, 1919.

During September, 1919, one case of typhus fever was reported in Nebraska.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS. City Reports for Week Ended Oct. 4, 1919.

•	Popula- tion as of July 1, 1917	Total deaths	Diph	t heria.	Mea	sles.		rlet ver.		ber- osis.
City.	(estimated by U. S. Census Bureau.)	from all causes.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths
berdeen, Wash bilene, Tex. dams, Mass kron, Ohto lameda, Calif. lbany, N. Y.	21,392						1			
bilene, Tex	21,392 14,954 14,406		1		1		5			
dams, Mass	14,406	2	3	•••••					;;-	
lemede Calif	93,604 28,433	25 0	3			¦•••••	3		18	
lhany N. Y	106,632	, ,	i				2		i	
	106,632 19,581	6	l		ĺ	
lton, Ill.	23.783	9		·			3			
iton, Ill nderson, Ind marbor, Mich miston, Ala msonia, Coun ppleton, Wis rington, Mass sbury Park, N. J shtabula, Ohio thanka, Ga	24,230	6								
min Arpor, Mich	15,041 14, 32 6	6	9						1	
reonia Conn	16,954	1	•				i		1	
poleton, Wis	18,005	5					l <u>-</u> .			
rlington, Mass	13,073	5 2 5 2					3			
sbury Park, N. J	14,629 22,008	5	1	1	····					ļ
shtabula, Ullo	22,008 196,144	2	12		2		7			Į
tlentic City N I	59,515	14	12				lí	i	1	1
ttlehoro Mass	59,515 19,776	3		i				l		
ustin, Tex	39,012	12	6	l						
akersfield, Calif	17,543	3		ļ						
altimore, Md	591,637	162	39	3	3		15		32	1
aton Rouge, La	17,544	1					2			
attle Crock, Mich	30, 159 72, 20 4	• • • • • • • •	6 8		1		5 1		2	
estrice Nebr	10, 437	3				•••••		•••••	1	
earmont. Tex	28,851	12			1				i	
edford, Ind	10,613	2	1							 .
eleit, Wis	18,547	2	1						1	
enton Harbor, Mich	11,099	.5				• • • • • •				1
erkeley, Calli	60, 427 13, 892	11 3	1		3	• • • • • •	3		1	
shtabula, Ohio. thantis, Ga. tlantic City, N. J. ttleboro, Mass. ustin, Tex. akersheld, Calif. altimore, Md. aton Rouge, La. attle Creek, Mich. ayonne, N. J. eatrice, Nebr. eatrice, Nebr. eatront, Tex. edford, Ind. eloit, Wis. enton Harbor, Mich. erkeley, Calif. erkely, Calif. ertin, N. H. everly, Mass.	22 128	3	• • • • • • • • • • • • • • • • • • • •				4		· · · · i	• • • •
everty, mass. iddeford, Me. irmingham, Ala huefield, W. Va. oise, Idaho.	17, 760 189, 716 16, 123 35, 951	ž					. :			ļ
irmingham, Ala	189,716	41	22				11		12	1
huefield, W. Va	16, 123		1				4			
oise, Idaho	35,951	3 170	78	4	19	• • • • • •	1 28		50	
oston, mass	767, 813	1/0	10	7	19		20		- 30	
oise, Idano	10, 472 124, 724 16, 318	26	18	i	11	i				
ristol, Conn	16,318	2	4				1			
rockton, Mass	69, 152 [9	1		6		2		2	
rookline, Mass	33,526	7	1			• • • • •			4	
runswick. Ga	10, 984 475, 781	108	142	10	• • • • • • • • • • • • • • • • • • • •	• • • • • •	18		23	
ullaio, N. I	25, 144	100	142	10	3	• • • • • •	10		23	
nrlington, Vt	21,802	5 7	1							
dillac, Mich	10, 158	4	3	1						
rockton, Mass ronswick, Ga. uffalo, N. Y. urlington, Iowa urlington Vt. sdillac, Mich siro, Ill	15,995	6	6							
ambridge, Mass	114, 293	29	6 7		1	· · · · · ·	3 6	• • • • • •	3	
imden, N. J	108, 117 62, 566	18	4			• • • • • •	1		3 2	
ape Girardeau, Mo.	11,146	10	4			•••••	•			İ
dar Rapids, Iowa	38,083		1							
nanute, Kans	12,968	3	1 2				3			
narleston, S. C	61,041	24	2 1			• • • • • •				
narieston, W. Va	31,060 40,759	9 16	4				3 5		3	
attenooge Tenn	61,575	16	7				2			
relsea. Mass	48,405	15	3		i				3	
neyenne, Wyo	1 11,320 2,547,201						4			
picago, Ill	2,547,201	524	157	18	28		102		195	
ncopee, Mass	29.960	2	• • • • • •				4		• • • • • •	
neimeti Ohio	15, 625	98	24		5	• • • • • • •	17	····i	14	• • • •
eveland. Ohio.	414, 248 692, 259	155	4	10	5 7		ii		16	
ape Girardeau, Mo. char Rapids, Iowa hanute, Kans. harleston, S. C. harleston, W. Va harleston, W. Va harleston, M. C. harleston, M. C. harleston, M. C. harleston, Mass. heyenne, Wyo hicago, Ill hicopee, Mass. hillicothe, Ohio hrcinnati, Ohio eveland, Ohio hiton, Iowa.	27,678	0								
inton, Mass	1 13 075	2							1	
inton Iowa. linton, Mass offeyville, Kans.	18, 331 25, 292		4				1		1	
DROCS, N. Y	25, 292 38, 965	4 14					• • • • • •		ii	
plorado Springs, Colo plumbia, S. C	38, 965 35, 165	14	2	•••••				• • • • • •	5 1	

¹ Population Apr. 15, 1910.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

	Popula- tion as of July 1, 1917	Total deaths	1 -	theria.	Mea	sles	Scr	arlet ver.	Tu	iber- losis.
City.	(estimated by U. S. Census Bureau).	from all causes.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Columbus, Ohio	220, 135	53	5	ļ			8	ļ	4	5
Corpus Christi, Tex	22,858 10,789 31,838	6 6								
Council Bluffs, Iowa	31,838	6	7				3			i
Corpus Christi, Tex. Council Bluffs, Iowa Covington, Ky Cranston, R. I.	59, 623	15 6	3				3			2
Cumberland, Md	26,773 26,686	4					2		2	
Cumberland, Md. Dallas, Tex Danvers, Mass Danville, Ill	129, 738 10, 037 32, 969	32	20	1			7			5
Danvers, Mass	10,037	4	1 2						25	
	20, 183		4						20	
Davenport, Iowa Davton, Ohio Dedham, Mass Denver, Colo Des Moines, Iowa	49,618		2							
Dayton, Ohio	128,939	29	6				3		6	1
Dednam, Mass	10,618 268,439	62	12	2	····i		····i			15
Des Moines, Iowa	104,052		. 8				14			1
Detroit, Mich	619,648	190	91	8	11		75	1	32	16
Detroit, Mich Dover, N H Dubuque, Iowa Duluth, Minn	13, 276	3	1 5							
Duluth. Minn	40,096 97,077	14	5		····i		2		2	i
	30, 286	6								Ī
East Cleveland, Ohio East Cleveland, Ohio Easthampton, Mass East Orange, N. J East St. Louis, III Eau Claire, Wis	13,864	i					2 1	• • • • • •		
Fast Orange N I	10,656 43,761	10	1 1		• • • • • •		1		4	·····i
East St. Louis, Ill.	77. 312	-8			2		i		3	
Eau Claire, Wis	18, 887						2			
Elgin, Ill	28, 562 38, 272	4 10	····i	····i	····i		• • • • • •			•••••
El Paso. Tex	69, 149	35	l							9
Elyria, Ohio	10 532	9			10					
Elmira, N. Y El Paso, Tex Elyria, Ohio Englewood, N. J Eureka, Cahif	12,603	1 3			•••••			• • • • • •		
Evanston III	15, 142 29, 304	13					1 1	•••••	• • • • • •	
Eureka, Cahr Evanston, Ill Everett, Mass Everett, Wash Fairmount, W. Va Fall River, Mass Fargo, N. Dak Findlay, Ohio Flint, Mich Fond du Lac, Wis Fort Scott, Kans. Fort Wayne, Ind	40.160	-6	5				4		3	
Everett, Wash	37, 205 16, 111	• • • • • • •	6				.4		• • • • • • •	
Fall River Mass	129, 828	i8	1		5		11		1	1
Fargo, N. Dak	17 872	3							. .	
Findlay, Ohio	14,858	5								
Fint, Mich	57, 386 21, 486	14 9	14	1	3		9			1
Fort Scott, Kans	10,564	3								
Fort Wayne, Ind	78 014	13	3				1		1	
Fort Worth, Tex	109, 597	23 5	32	2					1	1
Fort Wayne, Ind Fort Worth, Tex Fostorio, Ohio Framingham, Mass Freeport, Ill	10, 959 14, 149	1			••••					• • • • • •
Freeport, Ill.	19,844	5								
Fremont, Ohio	11,034	3 5			.					•••••
Galeshurg III	36,314	5	;		••••• •		2			1
Galesburg, Ill. Galveston, Tex Geneva, N. Y	24,629 42,650 13,915	7							i	
Geneva, N. Y.	13,915	6			-				<u>-</u> -	
Gloucester City, N. J. Grand Rapids, Mich.	11, 375 . 132, 861	28	14		2		2		3 5	•••••
Great Falls, Mont. Green Bay, Wis. Greenfield, Mass.	1 13, 948	10					2		il	····i
Green Bay, Wis	1 13, 948 30, 017 12, 251 20, 171	9		1 .						1
Greenfield, Mass	12, 251	5 5							•••••	• • • • •
Greenwich Conn	19, 594	3							····i	
Greenwich, Conn. Hackensack, N. J. Hammond, Ind. Hartford, Conn. Haverhill, Mass.	17, 412	5							1	•••••
Hammond, Ind	27,016 112,831 49,180	4	. 2	-			. 2		1	•••••;
Haverhill Mass	49 190	28 12	8 7		···i'		5	1	4	1
Hoboken, N. J.	78, 324	9	!		1:					3
Holland, Mich	12,459	4								
Holyoke, Mass	66.603 [10 31	2 2		1 -		2		2	• • • • •
Hudson, N. Y.	116, 878 12, 898	5	2	· · · · · · · · · · · ·						
Haverhill, Mass. Hoboken, N. J. Holland, Mich. Holyoke, Mass. Houston, Tex. Hudson, N. Y. Independence, Mo Indianapolis, Ind. Ironton, Oho	11,964	2								<u>.</u>
Indianapolis, Ind	283,622	69	10	1 .	-	•••••	9 .	•••••	8	7
попоп, ощо	14,079	7].		1 -	'-		' .		1.	•••••

¹ Population April 15, 1910.

DIPHTHERIA, MEASLES, SCABLET FEVER, AND TUBERCULOSIS—Continued.

	Popula- tion as of July 1, 1917	Total deaths	Diph	theria.	Mea	sles.		rlet ver.		ber- losis.
City.	(estimated by U. S. Census Bureau).	from all causes.	Cases.	Deaths.	Cases.	Daeths.	Cases.	Deaths.	Cases.	Deaths
haca, N. Y	16,017 37,431 14,411 312,557 10,678 50,408 14,270 192,096 305,816 24,325	5				J	1		2	
haca, N. Y	37,431	11	4		• • • • • •		2			
rsov City, N. J	312,557	1	13				2		24	1
hnstown, N. Yalamazoo, Mich	10,678	2	1							
alamazoo, Mich	50,408	14	3				1	ļ	3	1
ankakee, IH ansas City, Kans	192, 096	3	1 1				1		1 5	
ansas City, Mo.	305, 816	67	6		5		6		4	1
ansas City, Mo earny, N. J enosha, Wis.	24,325	' 4	1				2 2		1	
enosna, wis noxville, Tenn	32,833 59,112	3	1 6				3			
	21.929	10								1
ckawanna, N. Y	16, 219	7	4							
Crosse, Wis	31.833	7				• • • • •	1 2			
rayoue, IIII	21,481 16,086	5 2					11		11	
neaster, Ohiowrence, Kans	13,477	5					4		1	1
WIANCE MASS	1 0 2,923	15	2				7		4	
avenworth, Kansominster, Massxington, Ky	19.363							· · · · · ·	1	
vineton Kv	21,365 41,997	6 21	3		····i		3		1 22	
ma. Ohio	37, 145	9	$\frac{3}{2}$				i			
ma, Ohio neolm, Nebr	46,957 58,716 21,338	11	1				1			
tle Rock, Arkgansport, Ind	58,716	5	5		• • • • • •		1		4	
ng Beach, Calif	20 163 1	9					6		• • • • •	
ng Branch, N. J	15,733	2					i			
rain, Ohios Angeles, Calif	15,733 38,266 535,485	0					1			
s Angeles, Calif	535,485	101	12	2	2		8		34	
uisville, Kywell, Mass	240, 808 114, 366	66 27	26 3	2	1	· · · · · ·	6 5		7	
dington Mich	114,366 10,566 33,497	ï			i					
nchburg, Va	33,497	6								
nn Mace	104,534	13 10	11	2	1		6	• • • • • •	1	
dison, Wis	31,315 52,243	11					3			
	15,859	4					2		1	
nichester, N. H	79.607	22	4	1	ا نیز ۱۰۰۰	• • • • • •	1		1	
mitowoc, Wis	13,931 10,365 114,610	7 5	•••••		10	· · · · · ·	3	• • • • • •	1	
rinette. Wis.	1 14,610	i	i				1			
rion, ind	19.923	11	4				1			
rlboro, Mass	15, 285	6]				
rquette, Mich	12,555 12,984	4	4		•••••		2			• • • •
rtinsburg, W. Va. rtins Ferry, Ohioson City, Iowa	12,984 10,135	0								
son City, Iowa	14,938	6	2	1			6			
dford, Mass lrose, Mass	26,681	7 4					2		1	
mose, mass mphis. Tenn	17,724 151,877		16				7		8	
mphis, Tenn thuen, Mass	14,320	6								
ldletown, N. Yldletown, Ohio	15, 890 1		1	• • • • •	-		1		•••••	• • • •
ford, Mass	16,384 14,280	3 2	7		-				• • • • • •	• • • •
wankaa Wis	445,008	78	26	2			42		18	
neapolis, Minn	373,448 59,201	60	14	1	1 .		8		14	
bile, Alaline, Ill	59,201	23 14	6					• • • • •	····i	
ntelair, N. J.	27, 976 27, 087	0	3		····i·]:		3		î	
ntgomery, Ala	41,039	15	2] .		5			
rgantown, W. Va	14,444	1	1 .	· • • • • •	· · · · · · -				· · · · •	• • • •
ntgomery, Alargantown, W. Varristown, N. Jundsville, W. Va	13,410	4 2	5		-		1	•••••	• • • • • •	
	11,513 37,991	11	6						1	• • • •
shua, N. H.	37,991 27,541 118,136	6					3			
shua, N. H. shville, Tenn. wark, N. J.	118, 136	37	.5		1		3		3	
wark, N. J	418,789	90 30	19	····i	10 .		8 5		25 8	
w Bedford, Massw Britain, Conn	121,622 55,385 25,855	8	. .		1		3			
w Brunswick, N. J.	00,000	~ }	2		- 1				- 1	

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

	Popula- tion as of July 1, 1917	Total deaths	Dipl	itheria.	Mea	isles.		ariet ver.		ıber- losis.
City.	(estimated by U. S. Census Bureau).	from all causes.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Newburgh, N. Y	29, 893 15, 291 152, 275	7	2	ļ	ļ	 		i	2	. 2
Newburgh, N. Y. Newburyport, Mass. New Haven, Conn. New London, Conn. New Orleans, La. Newport, R. I. Newton, Mass. New York, N. Y. Niagara Falls, N. Y. Norfolk, Va.	152, 275	5 34 2	6	i	8		7		8	5
New London, Conn	21 100	2	1				1			.
New Orleans, La	377,010 30,585 44,345 5,737,492	93 8	11				5		17	11 2
Newton, Mass	44,345	6	4				l		1	1 1
New York, N. Y	5,737,492 38,466	1,103	162	9	31 3	3	31 1	1	247 3	110
Norfolk, Va	91,148		8				3		3	
Norfolk, Va. North Adams, Mass. North Attleboro, Mass. North Attleboro, Mass. North Little Rock, Ark North Tonowanda, N. Y. Norwalk, Conn. Norwich, Conn. Norwood, Ohio. Oakland, Calif. Oak Park Ill	91,148 122,019 20,006	4							1	
North Attlabora Mass	20,006 11,248	6 1	1			•••••				
North Little Rock, Ark	15 515	12	4							2
North Tonowanda, N. Y	14.060	6				•••••	2		····i	·····i
Norwich Conn	27,332 21,923		i			•••••				1
Norwood, Ohio	23, 269	3			4		1			
Oakland, Calif	206, 405	34 6	14	····i	2		6	• • • • • •	4	3
Oak Park, III Ogdensburg, N. Y Oklahoma City, Okla Olean, N. Y Omaha, Nebr	27,816 16,845 97,588 16,927	8								
Oklahoma City, Okla	97,588	16	3				1	•••••		1
Olean, N. Y	177,777	11 28	4	$\begin{vmatrix} 1\\1 \end{vmatrix}$		•••••	3	•••••		$\begin{bmatrix} & 1 \\ 2 \\ 2 \end{bmatrix}$
Orange, N. J.	33,636	5	2	<u>*</u> .			ıĭ			l
Orange, N. J. Oshkosh, Wis. Parkersburg, W. Va. Parsons, Kans.	36,549 21,059	6				•••••				1
Parkersburg, W. Va	15,952	4	4		;	•••••	····i		1	1
Pasadena, Calif	49,620 (6					1		3	2
Passaic, N. J	74,478	5 2	4			••••••	1		3	
Particlet R I	140,512	13	8			•••••	3		9	
Peekskill, N. Y	60,666 19,034	1								
Parsons, Kans Pasadena, Calif. Passaic, N. J. Paterson, N. J. Pawtucket, R. I. Peekskill, N. Y. Pekin, Ill	10,973	••••••	7	····i			2 12		• • • • • •	
Perth Amboy, N. J.	72,184 42,646	11	2	1			12		i	
Petria, III Perth Amboy, N. J. Petersburg, Va. Philadelphia, Pa. Phillipsburg, N. J.	25, 817	7	6						2	2
Philadelphia, Pa	1,735,514 15,879	341	91	5	21		31		77	31
Princ Bluff, Ark. Pittsfield, Mass. Platinfield, N. J. Plattsburgh, N. Y. Plymouth, Mass.	17,777		····2							
Pittsfield, Mass	39,678	9	• • • • •		;;-				••••	
Plattsburgh N. Y	24,330 13,111	6 7	i		11				2	
Plymouth, Mass	14,001	i 2	.					·		
Prymouth, Mass Pomona, Calif. Pontiac, Mich. Portland, Me. Portland, Oreg. Portsmouth, N. H.	13,624	21	15	····i	8				•••••	····i
Portland. Me.	18,006 64,720 308,399	22	10				3			3
Portland, Oreg	308, 399	48	5		1		8		1	2
Portsmouth, N. H	11,730 29,356	••••••	4 5			••••••	1 2		3	• • • • •
Portsmouth, Va	40,693	21	6				2			2
Poughkeepsie, N. Y	30, 786 259, 895	6 47	6	····i			2 2 8		1	5
Portsmouth, Ohio. Portsmouth, Va. Poughkeepsie, N. Y Providence, R. I Pueblo, Colo	56,084	ő	13 1	1			î l			
Quincy, Ill	36,832	7	1				1 2			
Quincy, Mass	39,022	9 12	6		1 .		3 3		3	·····i
Rahway, N. J	47, 465 10, 361	0								
Raleigh, N. C.	30, 274	6								•••••
Pueblo, Colo Quincy, Ill Quincy, Mass Racine, Wis. Rahway, N. J Raleigh, N. C Redlands, Calif Reno, Nev Richmond, Va Riverside, Calif Roanoke, Va. Rochester, N. Y.	14,573 15,514	0 2	•••••							
Richmond, Va	15, 514 158, 702 20, 496	56	18	i			10		25	3
Riverside, Calif	20,496	.8			.		····i	•••••		4
Rochester, N. Y.	46, 282 264, 714	10 51	3 29	3	3		12		24	
Rockford, Ill	56, 739	14	3				<u></u> .			
Rochester, N. Y. Rockford, Ill. Rock Island, Ill. Rocky Mount, N. C.	29, 452 12, 673	7 3	••••	-	•••••				1 1	•••••
LOURY MUULL, N. U		٥١	4						-	
Kome, Ga	10,007 1.		7:1							
Romé, Ga	15, 607 24, 259 68, 984	19	1				3		1	

¹ Population April 15, 1910.

DIPHTHERIA, MEASLES, SCARLET FEVER, AND TUBERCULOSIS—Continued.

	Popula- tion as of July 1, 1917	Total deaths	Diph	theria.	Mea	sles.		rlet ver.	Tu cul	ber- osis.
City.	(estimated by U. S. Census Bureau).	from all causes.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Saginaw, Mich St. Joseph, Mo St. Louis, Mo St. Paul, Minn	56, 469	20		<u>.</u> .	ļ <u>.</u>		<u>.</u> .	ļ		
St. Joseph, Mo	86,498 768,650	19 167	112	13	i		17		23	12
St. Paul, Minn	252, 565 49, 346	38	36	2	ļ <u>.</u> .		i		5	3
Salem, Mass.	49,346 121,623	10 31	4	1	····i	·····	4			3 2 2
San Bernardino, Calif	17,616	1 1								
Salem, Mass Salt Lake City, Utah Sen Bernardino, Calif. San Diago, Calif. Sandunty, Ohio. Sanford, Me.	17,616 56,412 20,226	17 2	1				····i			3
Sanford, Me.	11,217						. .			1
Sanart, Mo. San Trancisco, Calif. San Jose, Calif. Santa Barbara, Calif. Saugus, Mass. Savannah, Ga. Schenectady, N. Y. Seattle, Wash. Sloux City, Lowa. Sloux City, Lowa. Sloux Falls, S. Dak. Somerville, Mass. South Bend, Ind. Southbridge, Mass.	471,023	124	4	·	55		7 4		ļ	10
Santa Barbara, Calif	39,810 15,360 10,210 69,250 103,774	5					4		2	1
Saugus, Mass	10,210	2	2		 		·		ļ <u>.</u> .	
Schenectady, N. Y.	103, 774	36 11	6		i		5		3	3
Seattle, Wash	366, 445		7				12		ļ	
Signs Falls S Dak	58, 568 16, 887	3	3	1			3			
Somerville, Mass	88,618	19	1				6		4	2
South Bend, Ind	70,967	7 2							2	1
Spartansburg, S. C.	14,465 21.985	7	3			•••••				i
Spokane, Wash	21,985 167,656		5		2		3			
Springfield, Mass	62, 623 108, 668	18 32	1 2		• • • • • •	•••••	3 7		2	2 1
Springfield, Mo.	41,169	15								i
Springfield, Ohio	52, 29 6 31, 810	•••••	1 2			•••••			5	2
Steubenville, Ohio	28, 259 36, 209	6	1 2							
Stockton, Calif	36, 209	4	2				2			
Syracuse. N. Y	47, 167 158, 559	4 38	10				2 6	····i	4	i
Tacoma, Wash	117,446		7				ĭ			
Taunton, Mass	36, 610 67, 361	11 23	4			•••••			3	1 2
Tiffin, Ohio	67,361 12,962	4								
South Bend, Ind. Southbridge, Mass. Spartansburg, S. C. Spokans, Wash. Springfield, Mass. Springfield, Mass. Springfield, Mass. Springfield, Moss. Springfield, Moss. Springfield, Ohio. Stamford, Conn. Steubenville, Ohio. Stockton, Calif. Superior, Wis. Syracuse, N. Y. Tacoma, Wash. Taunton, Mass. Terre Haute, Ind. Tiffin, Ohio. Toledo, Ohio. Toledo, Ohio. Topeka, Kans. Trenton, N. J. Troy, N. Y. Tuscalooss, Ala. Vallejo, Calif.	202,010	50 12	6 2	1	5		10	1	4 5	1
Trenton, N. J.	49,538 113,974	25	2		i		i		6	
Troy, N. Y	75.104	30	2 2 2 7				2		5	
Valleio, Calif	10, 824 13, 803	3 5	7		•••••	•••••	1	•••••	1	
Vancouver, Wash	13,805		1							
Waltham Mass	34,015	15 3	4 3			•••••		•••••	•••••	
Washington, D.C	369, 282		18	2	···i		9		18	8
Waterbury, Conn	31,011 369,282 89,201 15,188	7	3				5		6	1
Watertown, N. Y.	30,404		2				2			
Wausau, Wis	19,666	5						•••••		1
Westfield, Mass	13,484 18,769 44,386	1 2	3				2		····i'	i
West Hoboken, N. J	44,386	2 5	3	1					2	
West New York, N. J	19,613 1 3,964	1	1			•		•••••	•••••	• • • • • •
Wheeling, W. Va	43,657	18	2							····i
White Plains, N. Y	23,331 73,597	8 14	3			•••••			12	1
Vallejo, Calif Vancouver, Wash Waco, Tex Watcham, Mass Washington, D. C. Waterbury, Conn Watertown, Mass Watertown, M. Y Wausan, Wis Webster, Mass Westfield, Mass West Hoboken, N. J West Orange, N. J West Orange, N. J Wheeling, W. Va White Plains, N. Y Wichita, Kans Wilmington, Del Wilmington, Del Wilmington, N. C. Winchester, Mass Winona, Minn Winton-Salem, N. C. Woburn, Mass Worcester, Mass Worcester, Mass Vonkers, N. Y	95,369	18-	8	:			2			1
Wilmington, N. C	30,400	14	5		1 .		1		1	1
Winona, Minn.	10, 812	4 8 7			5		1			
Winston-Salem, N. C.	1 18,583 33,136	7	4						2	i
Worcester, Mass	16,076 165,166	8 34	6				3	•••••	6	1 5
Worcester, Mass. Yonkers, N. Y	103, 066 112, 282	20	9		i		6		8	3
Youngstown, OhioZanesville, Ohio	112, 282 31, 320	21 7	5				7		8	4
	01,020	1	• • • • • • •	••••• •			• • • • • • [• • • • • • •	• • • • • •	•••••

¹ Population April 15, 1910.

FOREIGN.

AUSTRALIA.

Influenza-Mortality, July 12 to Aug. 15, 1919.

The accompanying table shows the reported mortality from influenza in Australia during the five-week period ended August 15, 1919. The table is taken from the Bulletin of the Quarantine Service of Australia, Vol. 4, No. 18, and is a continuation of the table which appeared in the Public Health Reports September 12, 1919, page 2093.

Number of deaths from influenza in Australia, July 12 to Aug. 15, by weeks.

	Vict	oria.	New Sou	th Wales.	South Australia.	Queens- land.	Western Australia.
Weck ended.	Whole State.	Metro- politan area.	Whole State.	Metro- politan area.	Whole State.	Metro- politan area.	Whole State.
July 18	120 135 162 155 76	102 92 109 67 37	540 343 231 117 120	150 103 82 41 56	8 9 8 15 18	1 2 3 2 0	13 5 30 26 41

ARGENTINA.

Destruction of Rats Obligatory.

According to information dated August 28, 1919, the extermination of rats has been made obligatory throughout the Republic of Argentina. All proprietors, tenants, or occupants of properties, vessels, railway properties or any other business premises are required to report the existence of epizoötic diseases in rats to the sanitary authorities and to lend their assistance of personnel, means, and resources required for strict compliance with the law. Infraction will be punished by fine.

BRAZIL.

Deaths from Communicable Diseases, July, 1919-Porto Alegre.

The following is a report of the deaths from communicable diseases in Porto Alegre, Rio Grande do Sul, Brazil, during the month of July, 1919. The population of Porto Alegre is said to be 150,000.

Causes of death.	Number of deaths.	Causes of death.	Number of deaths.
Bubonic plague	2 3 10	Measles Tuberculosis (all forms) Typhoid fever	1 53 4

CEYLON.

Influenza Mortality-January-March, 1919-Comparative Statistics.

Of the 58,522 total deaths registered in Ceylon during the first quarter of the year 1919, 12,324, or 21 per cent, were attributed to influenza. During the last quarter of 1918, the number of influenza deaths was stated to have been 18,887, or 28 per cent of the total deaths. Though the number of deaths throughout Ceylon during the first three months of 1919 showed a decrease from the number for the preceding quarter, the districts of Kurunegala, Puttalam, Chilaw, Batticaloa, and Anuradhapura showed considerable increases. The number of deaths from pneumonia in the period January to March, 1919, inclusive, was 2,834 (previous quarter, 12,556). The estimated population of the island of Ceylon on the last day of the quarter was 4,682,180.

Influenza and pneumonia in Ceylon-Number of deaths, October, 1918, to March, 1919.

	Fou	ırth quarter,	1918.	First quarter, 1919.				
District.	Influenza.	Pneumonia.	Combined.	Influenza.	Pneumonia.	Combined.		
Colombo Negombo Kalutara Kandy Matale Nuwara Eliya Galle Matara Hambantota Jaffua Mannar Mullaittivu Batticaloa Trincomalee Kurunegala Puttalam Chilaw Anuradhapura Badulla Ratnapura Ratnapura Kegalla Kegalla	1,093 1,234 1,099 619 1,095 607	925 15 544 2,994 2,059 353 353 127 69 516 80 18 351 60 47 95 1,366 722 1,163	2, 810 353 1, 637 4, 228 2, 080 2, 678 1, 448 1, 103 2, 558 342 338 323 2, 178 92 95 934 3, 762 1, 338 1, 934	801 299 66 393 105 163 146 140 424 403 41 599 1, 208 123 4, 632 252 214 1, 419 1, 165 122 149	323 29 45 504 111 273 13 26 20 250 28 42 98 42 242 242 59 777 59 300 260 33	1, 124 325 111 897 216 433 155 166 444 653 69 101 1, 306 185 4, 874 311 291 1, 478 322 1, 465		
Ceylon	18, 887	12,556	31,443	12, 324	2, 834	15, 158		

Influenza and pneumonia in Ceylon—Annual death rates per 100,000 population, October, 1918, to March, 1919.

	Fou	rth quarter,	1918.	First quarter, 1919.				
District.	Influenza.	Pneumonis.	Combined.	Influenza.	Pneumonia.	Combined		
Colombo	1,063	522	1,584	462	186	64		
Negombo	703	32	734	635	62	69		
Kalutara		694	2,087	86	59	14		
Kandy	1,114	2,704	3,818	363	465	82		
Matale	4, 034	3,600	7,634	394	416	81		
Nuwara Eliya	1,448	4,816	6, 264	390	653	1,04		
Galle	1, 342 939	433	1,775	183	16	19		
fatara	939	197	1,136	222	41	26		
Hambantota	3,646	243	3,889	1,528	72	1,60		
affna	2,371	599	2,970	478	297	77		
Mannar	7,485	422	7,907	708	484	1,19		
Mullaittivu	6, 791	1,055	7,846	1,384	985	2,36		
Batticaloa	617	192	809	2, 955	240	3,19		
Crincomalee	3, 941	233	4,173	1, 625	555	2, 17		
Kurunegala	2,278	438	2,716	5, 904	308	6, 21		
Puttalam	328	616	945	2,645	619	3, 26		
hilaw	196	192	389	895	322	1,21		
Anuradhapura	3, 793	429	4,222	6,558	273	6, 83		
	4,271	2, 435	6,706	2,123	547	2,67		
Ratnapura	1,420	1,664	3,085	288	C13	90		
Kegalia	1, 209	1,801	3,010	236	52	28		
Ceylon	1,599	1,063	2,662	1,067	245	1,31		

CUBA. Communicable Diseases—Habana.

Communicable diseases have been notified at Habana as follows:

	Aug. 1	0–31, 1919.	Sept.	Remain-	
Disease.	New cases.	Deaths.	New cases.	Deaths.	treatment Sept. 30, 1919.
Broncho-pneumonia. Chickenpox. Diphtheria. Influenza	1 4 3	1 2	10 3 5 5	9 1 1	4 1 4 18
Malaria Measles Paratyphoid fever Scarlet fever Smallpox Typhoid fever	89 7 . 3 . 1	1	129 2 1 1 2 41	3	158 5 2 2 2 2 24 368

¹ From the interior 26.

INDIA.

Influenza-Coorg Province-1918.

A report by Lieut. Col. E. Hasell Wright, I. M. S., Civil Surgeon, Coorg, published in the Indian Medical Gazette for August, 1919, states that epidemic influenza was reported from South Coorg in June, 1918. The first wave, which attacked different parts of the province at different times from June to August, was of a mild

² From abroad 1.

From the interior 25; from S. S. Venezia 1.

nature, not attended with a high rate of mortality, and lasted only a few weeks. It was followed by a severe wave, the disease spreading rapidly and causing a high mortality.

The total number of deaths in Coorg up to the end of November, 1918, was 3,382. The population in 1911 was 174,976, giving a death rate of 19.3 per 1,000 population.

Influenza-Rangoon-June 1 to August 16, 1919.

The following table gives the number of deaths from influenza in Rangoon, British Burma, India, during the period from June 1 to August 16, 1919. The population of Rangoon is estimated at 326,244. The death rate for the 11 weeks was 13.3 per 1,000 population per annum, and the annual death rate for the "peak" week (ended July 12) was 18.4 per 1,000.

Week ended—	Number of deaths.	Week ended—	Number of deaths.
June 7. June 14. June 21. June 28. July 5. July 12. July 19.	19 30 66 107 92 115 108	July 26. Aug. 2. Aug. 9. Aug. 16. Total.	102 94

PARAGUAY.

Plague.

A report dated October 13, 1919, states that a number of cases of bubonic plague have developed at Asuncion, Paraguay, with three deaths. It is also reported that the disease exists in several river ports between Asuncion and Buenos Aires.

PHILIPPINE ISLANDS.

Cholera-Manila and Provinces-1914-1919.

The tables following give a record of cholera in Manila for the years 1914 to 1918 and part of 1919, and in the Provinces of the Philippine Islands for the years 1917 and 1918 and the year 1919 up to and including the month of July.

Cholera in Manila—Cases and deaths, by weeks, for the years 1914 to 1918, and part of 1919.

Wash and ad		1919	1	918	1	917	1	1916	1	1915	1	1914
Week ended.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
Jan. 4	4 6	3.	0	0	3	1 0	1 2	1	4 2 3	2 2	4 8 11	2 7
18 25 Feb. 1 8	4 2 6 3 0	3° 1 3 3 1 0	1 0 0	0 0 0	1 3 6 0	1 0 3 0	1 4 7 3	0 3 4 0	10 2 0	2 2 7 1 0	6 8 2	5 0
15 22 Mar. 1 8	0 1 0 3	0	0 0 0	0	5 1 0 1	2 0 0	3 2 0 2 3	2 0 1 0	3 8 5 7	1 6 3 4	6 3 0 3	2 76 11 50 33 00 22 00
15 22 29 Apr. 5	0 3 1 1 4	0 0 4 4	0	0	1 0 0	1 0 0	1 0 0	0 0 0	8 8 6 0	5 4 4 0	2 0 0	0
12 19 26 May 3	9 4 3 0 2 2 1	1 2 0 1	0	0	0 0 1 0	0 0 1	0 1 2 3	0 1 2 3	2 1 0	2 1 0	0	0 0 0
10 17 24	2 1 3 1	1 0 1 0	0	0	0	0	5 12 17 12	12 5 2	0 0 0	0	0	0 0 0
June 7	0 0 2	0 0 1	0	0	0 0 1	0	9 7 8	1 6 4	0	0	0	0
July 5 12 19	4 14 40 45	3 10 18 25	0	0	0	0 0 0	6 9 . 14 15	2 3 4 6	0 0 2 0	0 0 1 0	0 3 14 6	0 0 3 13 3 9
Aug. 26 9 16	67 95 71 74	33 37 47 44	0	0	0 0 2	0 0 0	26 22 37 41	13 12 14 25	0 0 1	0 0 1	10 39 23 14	9 31 9 14
23 30 Sept. 6			0	0	3 0 1 0	0	112 115 123 88	56 45 68 40	0	0	21 22 58 39	12 11 18 20
20 27 Oct. 4			0 5 23 31	0 4 19 21	0 0	0 0	104 62 41 22	36 25 17	0 0	0	89 23 22 13	47 8 10 9
18 25 Vov. 1			13 15 20	9 17 12	0	0	27 5 9	10 4 1	0	0	7 3 5	4 0 2 1
8 15 22 29			8 28 12 6	14 10 2	0	0 0 0	7 6 2 31	0 2 1 10	0	0	1 8 1 0	3 1 0
Dec. 6			7 7 4 1	4 3 1 1	0	0	141 43 3 8	40 12 4 2	0 0 0	0 0 0	19 7 6 2	10 5 1

Cholera in the Philippine Provinces, excluding Manila—Cases and deaths, by months, for the years 1917, 1918, and part of 1919.

W 41	19)17	19	918	1919	
Months.	Cases.	Deaths.	Cases.	Deaths.	Cases.	Deaths.
January	839	594	475	354	550	391
February	1,068	720	536	441	338	224
March	842	510	289	218	287	214
April	424	223	172	118	200	165
May	840	497	176	89	573	395
June	1,390	814	226	140	945	647
July	2,149	1,319	379	220	5,837	4,430
August	1,627	956	620	362		
September	239	513	384	269	l	
October	819	487	989	725		
November	1,278	787	1,203	1,049		
December	837	531	791	620		

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER. Reports Received During Week Ended Oct. 24, 1919.1

CHOLERA.

Place.	Date.	Cases.	Deaths.	Remarks.
Ceylon:	Tulm 0f			Outhord 140 miles from Co
Hambantota	July 25			Outbreak 148 miles from Co- lombo. Spread to other places.
China: Antung	Sent 1-7	238	68	
Chefoo	Sept. 1-7	200		Daily average over 50 fatalities.
Peking Tientsin	Aug. 24–30 Aug. 24–Sept. 6	68	1	Foreign. Native city.
India:	i		1	
Bombay	Aug. 3–9 Aug. 18–Sept. 6	29 17	22 11	
Mesopotamia: Bassorah	July 20-26	1		
Philippine Islands:	_			
Manila Provinces	Aug. 17-23	71	28	Aug. 17-23, 1919; Cases, 2,075;
Provinces. Ambos Camarines. Batangas Bohol Bulacan	Aug. 17-23	28	24	deaths, 1,518.
Batangas Bohol	do	101	71 2	
Bulacan	do	26	20	
Ilocos Norte	do	301	176 2	
Ilocos Sur	do	156	102 28	
Laguna	do	41 70	48	
Misamis	do	5 41	4 34	
Oriental Negros	do	34	23	
Pampanga	do	15 1, 153	13 500	
Rizal	do	50	34	
Tayabas Union	do	4 48	6 31	
Bulacan. Cebu. Hocos Norte. Hocos Sur Hoilo. Laguna. Misamis. Nueva Ecija. Oriental Negros. Pampanga. Pangasinan. Rizal. Tayabas. Union. Manila. Provinces.	Aug. 24-30	69	27	Ann 04 20 1010: Cones 1 1414
Bataan	Aug. 24-30	1		Aug. 24-30, 1919: Cases, 1,141; deaths, 801.
Union Manila Provinces Bataan Batangas Bohol Bulacan Capiz Cavite Cebu Hocos Norte Hocos Sur Hollo Laguna Leyte Mindoro Nueva Ecija Occidental Negros Oriental Negros Pampanga Pangasinan Tayabas Union Manila Provinces	do	125	80	•
Bulacan	do	8 9	4	
Capiz	do	11 68	6 44	
Cebu.	do	226	153	
Ilocos Norte	do	49 55	41 44	
Iloilo	do	43	25	
Laguna Leyte	do	41 41	36 18	
Mindoro	do	79	45 7	
Occidental Negros	dio	9 14	11	
Oriental Negros	do	20 4	12	
Pangasinan	do	207	166	
Union.	do	12 119	9 92	
Manila	Aug. 31-Sept. 6	45	22	Aug. 31-Sept. 6, 1919; Cases,
Provinces Albay Ambos Camarines Bataan Batangas Bohol Bulgeon	Aug. 31-Sept. 6		i	1,572; deaths, 1,098.
Ambos Camarines	do	120	86	
Batangas	do	56	42	
Bohol	do	13 15	7 13	
Capiz	do	10	7]	
Cebu	do	18 164	11 95	
liocos Norte	do	71 65	52 49	
Bohol Rulacan Capiz Cavite Cebu Hocos Norte Hoilo Laguna Mountain Province Nueva Ecija Occidental Negros Oriental Negros Pampanga Pangasinan Rizal	do	44	33	
Mountain Province	do	37	26	
Occidental Negros	do	52	37	
Pampanga	do	7 3	8 3	•
Pangasinan	do	505	354	
Kizai	ao	184	115 1	

¹ From medical officers of the Public Health Service, American consuls, and other sources.

Reports Received During Week Ended Oct. 24, 1919—Continued.

CHOLERA—Continued.

India.	Place.	Date.	Cases.	Deaths.	Remarks.					
Azores: Fayal Island	Provinces—Continued. Tayabas Union. Zambales Siam:	dodododo	157	127 115						
Fayal Island Sept. 6-19		PLA	GUE.	1						
Fayal Island Sept. 6-19	A zoros:			1						
Island Colombo Colombo Aug. 10-16. 2 2 2 2 2 2 2 2 2	Fayal Island Terceira Island British East Africa:	do			Do.					
Colombo		Aug. 3-16	••••••		Present in vicinity. Zanzibar Island.					
Total number of cases reported Aug. 27, 11; deaths, 3.	_ Colombo	Aug. 10–16	2	2	Town cases.					
India	Alexandria France:	Sept. 3-9	3	2						
Bombay					Total number of cases reported to Aug. 27, 11; deaths, 3. Aug. 3-9, 1919: Cases, 547; deaths,					
Bagdad	Karachi Madras-Presidency	Aug. 18-23	11	9	413.					
Aug. 19	Bagdad Bassorah. Syria:	1		i ,						
Mar. 25; arrived Buenos Air May 9; sailed June 20; arrived St. Vincent, Car Werde Islands, July 10.	On vessel:		••••••		•					
Cānada: New Brunswick— Antigonish County Sept. 28-Oct. 4 Present. Egypt: Alexandria Sept. 3-9 9 3 India: Bombay Aug. 3-9 11 4 Madras Aug. 18-Sept. 6 54 26	S. S. Clan Dailloit	Aug. 18	•	·	Mar. 23; arrived Buenos Aires May 9; sailed June 20; arrived Montevideo and sailed June 21; arrived St. Vincent. Cape					
New Brunswick— Antigonish County Sept. 28-Oct. 4 Present. Egypt: Alexandria. Sept. 3-9 9 3 India: Bombay Aug. 3-9 11 4 Madras Aug. 18-Sept. 6 54 26	-	- SMALLPOX.								
Antigonish County Sept. 28–Oct. 4 Present. Egypt:	Cănada:									
Alexandria. Sept. 3-9 9 3 India: Bombay Aug. 3-9 11 4 Madras. Aug. 18-Sept. 6 54 26	Antigonish County	Sept. 28-Oct. 4			Present.					
Magras	Alexandria	- I	9	3						
	Madras	Aug. 3-9 Aug. 18-Sept. 6		26 26						
Spain:	San Luis Potosi	Sept. 28-Oct. 4		1						
Malaga. Aug. 1-31 1 Valencia. Aug. 31-Sept. 6 9 2 Vigo. Sept. 7-20 2	Malaga	Aug. 31–Sept. 6 Sept. 7–20	9	2						
TYPHUS FEVER.										
Egypt: Alexandria	Alexandria	Sept. 3-9	11	5						
Italy:	Venice	Sept. 8-14	13	1						
San Luis Potosi Sept. 28-Oct. 4 Present. Sumatra: Medan July 27-Aug. 2 1	San Luis Potosi Sumatra:	- 1	1		Present.					

Reports Received from June 28 to Oct. 17, 1919.

CHOLERA.

Ceylon: Colomba	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
China: Amoy	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
Amoy	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
Foochow July 10-26 To July 16 Average of 1 ties daily. To July 26: of 30 cases daily. To July 28: pidemic.	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
Foochow July 10-26 To July 16 Average of 1 ties daily. To July 26: of 30 cases daily. To July 28: pidemic.	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
Foochow July 10-26 To July 16 Average of 1 ties daily. To July 26: of 30 cases daily. To July 28: pidemic.	00 fatali- Average ive fatal 27-Aug. ent from igh mor- of physi- conces- Deaths
Foochow	00 fatali Average ive fatal 27-Aug ent from igh mor- of physi- conces- Deaths
Foochow	ent from igh mor- of physi- conces- Deaths
Hongkong	of physiconces- Deaths
Shanghai	of physiconces- Deaths
Swatow	of physic concess Deaths
Do. June 29-Aug. 9 115	conces- Deaths
Tsingtao. July 6-Aug. 31. 59 Ungkung. Aug. 16	conces- Deaths
Ungkung	ession.
Chosen (Korea). Aug. 15. 3 Aug. 26: 6 cases. Anyo. do. 1 Keiki Province. New Wiju. Aug. 12. 1 In a Korean arrived fr Seoul. Aug. 15. 1 was prevalent. Shingshu. do. 1 North Heian Province. South Kankyo Aug. 26: 6 cases. In a Korean arrived fr tung, China, where was prevalent. North Heian Province. Present. South Kankyo Aug. 26: 6 cases. In a Korean arrived fr tung, China, where was prevalent. North Heian Province. Present. South Kankyo Aug. 26: 6 cases. In a Korean arrived fr tung, China, where was prevalent. North Heian Province. Present. South Kankyo In Bound Present. South Kankyo Aug. 26: 6 cases. In a Korean arrived fr tung, China, where was prevalent. North Heian Province. Present. South Kankyo In Bound Present. South Kankyo Aug. 28: 08: 45: 55: 56: 56: 56: 56: 56: 56: 56: 56: 5	
Anyo	watow.
Seoul. Aug. 15. 1 was prevalent.	
Seoul. Aug. 15. 1 was prevalent.	
South Kankyo	cholera
South Kankyo	
Bombay Apr. 28-June 28 84 55 Do June 29-Aug. 2 128 69 Calcutta May 4-June 21 617 Do June 29-Aug. 9 100 Karachi July 24-30 3 2 Madras May 18-June 28 29 19 Jan. 19-25, 1919; Case Do July 12-Aug. 16 33 18 deaths, 75. Rangoon Apr. 28-June 28 108 85	
Calcutta. May 4-June 21. 617 Do. June 29-Aug. 9. 100 Karachi. July 24-30. 3 Madras. May 18-June 28. 29 19 Do. July 12-Aug. 16. 33 18 Rangoon. Apr. 28-June 28. 108 85	
Calcutta.	
Karachi July 24-30 3 2 Madras May 18-June 28 29 19 Jan. 19-25, 1919; Cast Do July 12-Aug. 16 33 18 deaths, 75. Rangoon Apr. 28-June 28 108 85	
Madras. May 18-June 28. 29 19 Jan. 19-25, 1919: Case Do. July 12-Aug. 16. 33 18 deaths, 75. Rangoon. Apr. 28-June 28. 108 85	
Do	oe 113•
Rangoon Apr. 28-June 28 108 85	, IIO,
170	
Indo-China: Cochin-China—	
Saigon Apr. 21-June 29 386 272 City and district.	
Do	
Japan:	
Pascadores Islands July 14 49 In one village.	ooo 308•
Taiwan Island July 2-Aug. 12, 1919: Ca. deaths, 245.	sca, 000,
Keeling Aug. 8. Present in vicinity.	
Taihoku do Present.	
Tokyo	
Seet Java	ses. 613:
East Java Apr. 23-June 20 97 79 deaths, 507. June 25- Do June 25-July 15. 15 13 lg19: Cases, 16; deaths Mid-Java Bara May 20, 1919: Cases	July 15.
Surabaya Apr. 23-June 20 97 79 deaths, 507. June 25-Dune 25-Duly 15 Do June 25-July 15 15 13 1919: Cases, 16; deaths	, 18.
Mid-Java. Mar. 28-May 30. 89 84 deaths, 1,525.	s, 1,914;
West Java May 2-June 26, 1919: Cases, 10, Do. Aug. 2-8. 1 1919: Cases, 10; deaths, 67. July 18-1	Aug. 2.
Do	5.
Manchuria: Darien	
Persia: May 2. Present.	unding idemic, nber of
Ardebil May 2 Present. Enzeli Apr. 23 1	ninding idemic, nber of
Khorram-Ahah May 3	unding idemic, nber of
	unding idemic, nber of
Zindjan Apr. 21-May 4 49	unding idemic, nber of

Reports Received from June 28 to Oct. 17, 1919-Continued.

CHOLERA—Continued.

Place.	Date.	Cases.	. Deaths.	Remarks.
Philippine Islands:			1	
Manila Do	Apr. 26-June 28 June 29-Aug. 2	11 261		
ProvincesBantangas	May 4-24.	25	23	May 4-24, 1919: Cases, 567; deaths
Bulacan	do	48	25	
Cebu	t do	: 20		
Mindoro	do	19	14	
Pampanag	do	166		1
Tavahas		118		
Provinces Bantangas	June 1-28	79	61	June 1-28, 1919: Cases, 615: death:
Bohol	June 15-28	11	8	
Bulacan Cavite	June 1-28	63 23	27 14	1
Cebu	June 22-28	24	11	1
Laguna Hocos Sur	June 8-21	16	13	1
Nueva Ecija	June 1-28	60		1
Pampanga Pangasinan	June 8-28	105 113	79 81	
Tayabas	do	108	81	·
Union Provinces	June 22-28	7	7	June 29-Aug. 16, 1919: Cases, 8,255
Ambos Camarines	July 27-Aug, 16	117	66	deaths, 5,914.
Bataan	July 6-Aug. 9	668	536	
Bantangas	June 29-Aug. 16 do	31	24	
Bulacan	do	435	320 115	·
Cavite	June 29–July 26	174 84	41	
Cebu	Aug. 10-16	3	120	
Ilocos SurIloilo	June 29-July 26 Aug. 10-16 July 20-Aug. 9 July 6-Aug. 16	177 33	23	
Laguna		164	121	
Mindoro	July 20-26do	125 6	45 4	
Mountain	July 6-12	9	2	
Neueva Ecija Oriental Negros	June 29-Aug. 16 July 27-Aug. 16	424 112	298 60	
Pampanga	June 27-Aug. 16	546	441	
Pangasinan Rizal	July 13-Aug. 16	3,962 370	2,843 228	
Sorsogon	July 27-Aug. 16]	23	21	
Tayabas Union	June 29-Aug, 16 July 6-Aug, 16	329 402	266 291	•
Zambales	July 6-Aug. 16 July 13-19	1	1	
iam: Bangkok	Apr. 12-June 28		697 28	
Dotraits Settlements:	June 30-Aug. 2	• • • • • • • • •		
Singapore	July 14-27	80	69	Sept. 30: Present.
umatra: Medan	June 29-July 13-26.	8	1	Present in neighboring villages, June-July, 1919.
urkey: Constantinople	July 28			Present.
n vessel:		1		At Yokohama, from Shanghai,
Steamship	Aug. 17	1		Aug. 12, 1919.
·	PLAC	GUE.		
razil:	1			
Ceara.	Sept. 8			Present.
Pernambuco	May 28-June 1	• • • • • • • •	1	
			i	Th
ritish East Africa: Kisumu	May 18-June 28			Present. Zanzibar Island.
Kisumu	May 18-June 28 June 29-July 26 June 15-21	1		Do.

Reports Received from June 28 to Oct. 17, 1919—Continued.

PLAGUE-Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
China:		,		,
	June 17-23		1	
Amoy Do	Aug. 18–25 May 25–June 28		1	1
Canton	May 25-June 28			Present. Apr. 27-May 10, 1919 Cases, 3;present May 24-June 7
Foochow	May 18-24	 	l	1919. Present.
Hongkong	June 15-28	42	33	i
Do Ecuador:	June 29-Aug. 9	35	31	
Guayaquil Posorja	June 16-30 June 1-30	2 3	1	Bathing place 65 kilometers from
EgyptCities—	······································			Guayaquil. Jan. 1-Aug. 6, 1919: Cases, 740 deaths, 405.
Alexandria	July 23-29	1		doubles, 100.
Ismarlia	July 23-29 July 29	2		
Cairo Kantarah	May 1	[1	
Kantarah	June 19-20	4	2	Two European. Septicemic.
Do	July 31-Aug. 3	2	3	
Port Said	July 31-Aug. 3 May 1-June 28 July 4-Aug. 2	9	10	
Do	July 4-Aug. 2	2	4	
Suez Provinces—	June 5-11	3	3	
Assiout	May 17-June 24	80	41	
Do	July 3-Aug. 6	7	3	
Beni-Souef	July 3-Aug. 6 May 19-June 21 May 18-July 5 May 15-July 8	6 10	5 7	
FayoumGirgeh	May 15_July 8	32	10	
Menousia	June 8–24	5	10	
Menoufia	May 24-June 25	29	11	•
Do	June 8-24 May 24-June 25 July 5-7	3	1	
Marseille	Aug. 16-Sept. 2	. 5	3	
Liverpool	July 30	1	1	In dock laborer.
Ah Poi Camp	Aug. 9.	1	1	
Poouhau	July 19	ī	. .	
Kukuiau	Sept. 23	3	3	
Kukuiau Paauilo	Sept. 25	2	1	
ndia				Apr. 27-June 28, 1919: Cases, 8,645
Bombay	Apr. 28-June 28 June 29-Aug. 2 May 18-June 14	278	202	deaths, 6,933. June 29-Aug. 2 1919: Cases, 2,076; deaths, 1,561
0 Do	June 29-Aug. 2	32	19	1919: Cases, 2,076; deaths, 1,361
Calcutta	May 18-June 14	••••	38 22	
Karachi.	June 28-Aug. 2 May 18-June 28	145	132	
Do	June 29-Aug. 9	42	39	
Do	Aug. 28-30	5	4	
Madras				Jan. 19-25, 1919: Cases, 2; deaths,
i	Tul- 0 A 10	001	237	1. Jan. 19–25, 1919: Cases, 586
Madras Presidency Rangoon	July 6-Aug. 16 Apr. 28-June 28	381 75	63	deaths 347 May 30-June 5
Do	July 6-Aug. 16	158	144	deaths, 347. May 30-June 5. Cases, 37; deaths, 28.
ndo-China:	our, o mag. 10	100		
Cochin China—				
Saigon	Apr. 21-June 29	31	23 3	City and district.
Do	July 28-Aug. 10	4	3	-
ipan: Yokohama	June 9-15	1	1	
ava:		- 1	- 1	
East Java	Apr. 23-June 3	7	7	Apr. 8-June 28, 1919: Cases, 130; deaths, 130. July 23-29, 1919: Cases, 34; deaths, 34. Apr. 26-May 30, 1919: Cases, 23;
		1	1	Cases, 34; deaths, 34.
	Apr. 26-May 20	10	10	deaths, 23.
Mid-JavaSamarang		240	269	
Samaranglesopotamia:	- 1			
Samarangfesopotamia:	Apr. 19-June 20	346	11	
Samarangfesopotamia:	- 1	108	89	Total from data of officience.
Samarang	Apr. 19-June 20	2	89	Including suburb of Ashar. Total from date of outbreak, March, 1919, to June 24, 1199: Cases, 396; deaths, 256.
Samarang	Apr. 19-June 20	2	2	Total from data of officers.

Reports Received from June 28 to Oct. 17, 1919—Continued.

PLAGUE—Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Straits Settlements: Singapore Do. Turkey:	Apr. 13-26	2 11	1 7	
ConstantinopleOn vessel: S. S. City of Sparta	Oct. 9	1	1	Bubonic and pneumonic. From Bombay, Apr. 3, 1919: Case,
Do	May 13-17	1	1	a soldier at sea. At Liverpool: Case, a native member of the crew. (Public Health Reports, June 27, 1919.
S. S. Framlington Court	July 25	. 1		p. 1463.) From Alexandria, May 30; from Montreal, July 4; from Sydney, Nova Scotia, July 9; at Avon- mouth, England, July 22, 1919.

SMALLPOX.

				4
Algeria:	l	ļ.	i	
Algiers	June 1-30	1	1	
Do	July 1-Aug. 31		5	
Arabia:	July 1-11ug. 01	1 -0		
Aden	May 13-19	ı	1	1
Austria	1 1143 10-15:		1 *	Mar. 9-Apr. 5, 1919: Cases, 92.
Salzburg	Mar. 9-Apr. 5	50		Blat. 5-Apr. 0, 1919. Cases, 92.
Vienna	do	17	1	
Azores:		1		Į.
St. Michaels	June 7-20	1	-	4
Brazil:	June 1-20]
Bahia	Apr. 20-June 7	4	I	1
Pernambuco	May 4-25	5		Tom 1 Mom 2 1010: Come 10
Rio de Janeiro	May 11-June 21	61	20	Jan. 1-May 3, 1919: Cases, 10.
Do British East Africa:	June 30-Aug. 9	148	46	į.
British East Airicu:	Man 0 0	1		g1
Kisumu	Mar. 2-8		1	Zanzibar Island.
Mombasa	Mar. 1-June 7		37	D
Mtebba	Mar. 24-Apr. 6			Present. In Uganda.
Nairobi	Mar. 1-May 31	3		
Prison Island Quarantine		1	1	Zanzibar Island. In February,
Station.	!		ļ.	1919. From vessel from India.
Canada:		l	[
British Columbia—	l	_	l	
Vancouver	June 15-Sept. 11	8		
New Brunswick—			1	
Campbellton	June 15-21	1		•
Ďo	AugSept. 6	1		
Gloucester County	.			July 1-Sept. 30, 1919: Cases, 12.
Moneton	July 6-12	1		
St. John	July 27-Aug. 2	1		
Nova Scotia—				
Cities—				
Bridgenorth	July 27-Aug. 9	• • • • • • • •		A few cases; mild.
Halifax	June 28-Sept. 20	65		June 15-28, 1919: Cases, 82.
Sydney	June 8-21	3		•
Do	Aug. 1-Sept. 6	4		
Counties—				
Antigonish	June 28-Sept. 6			Present.
Colchester	Aug. 3-8			Do.
Cumberland	Aug. 30-Sept. 6			Do.
Guysborough	Aug. 18-30			Do.
Do	Aug. 18-30 Sept. 21-27			Do.
Halifax	June 28-Sept. 27			Do.
Hants	do			Do.
Kings	Aug. 10-16			Do.
Lunenburg	July 13-Aug. 16			Do.
Pictou	July 20-Sept. 13			Present. Also on Cape Breton
	, 20 copt. 10			Island, July 27-Aug. 21.
Richmond	Aug. 24-Sept. 20		1	Present.
Shelbourne	Aug. 24-30			Do.
Victoria	Aug. 3-9	• • • • • • • • • • • • • • • • • • • •		Do.
***************************************	1.46. 0-0	• • • • • • • • • •		<i>D</i> v.

Reports Received from June 28 to Oct. 17, 1919—Continued.

SMALLPOX-Continued.

· Place.	Date.	Cases.	Deaths.	Remarks.
Canada—Continued.				
Ontario—		1	1	ł
Province	.		.	May 1-June 30, 1919: Cases, 166 deaths, 4. July, 1-31, 1919 Cases, 51; deaths, 1.
***	T M 4 0	١ .	1	deaths, 4. July, 1-31, 1919
Hamilton	June 29-Aug. 2	2 14		Cases, 51; deaths, 1.
Harwich North Bay	May 1-31 Sept. 21-27	l i		Township in Kent County.
Ottawa	June 15-21	2		
Do	June 15-21. June 29-Sept. 6 June 15-21.	3		
Do	June 15-21	4		
Toronto		1		
Do Walpole Island	May 1-31	42		Kent Coutny. Island in Lake
Prince Edward Island-	May 1-01	1 22		St. Clair. Among Indians.
Charlottetown	July 16-Aug. 9	8		St. Camer. Millions Indians.
Quebec				In Bonaventure and Gaspe
	1			Counties, Aug. 1-31, 1919:
Montreal	June 8-28	18 11		cases.
Do Quebec	Aug. 24-Sept. 27	18		June 8-14, 1919: 1 case on incom
Do	June 8-28 July 5-Sept. 20	41		ing vessel.
Restigouche	June 15-July 31	40		Estimated. On Indian reserve.
Ceylon:			1	
Colombo	May 1-31	4 3	1	June 17-23: Present.
Do	July 13-Aug. 23		1	
China: Amoy	May 23-June 16. July 8-21 July 29-Aug. 25. May 18-June 21. July 1-Aug. 16. June 8-21. May 4-June 28. June 29-Aug. 23. May 18-June 29. May 18-June 29. June 29-June 29. June 29-June 29. June 29-June 29. June 29-Aug. 30.		13	
Do	July 8-21			Present.
Do	July 29-Aug. 25			Do.
Canton	May 18-June 21			Do.
Do	July 1-Aug. 16			Do.
Chefoo	June 8-21			Do. Do.
Chungking Do	Tuno 20 Apr 22			Do. Do.
Foochow	May 18-Aug. 23			Do.
Honekong	May 18-June 28	5	5	Do.
Hongkong. Nanking.	May 25-June 28			Do.
. Do	June 29-Aug. 30			Do.
Chosen (Korea):	Apr. 1-June 30	22	1 4	
Chemulpo Do	July 1-31	ī	ī	
Filean	do	336	96	
Do	do	4		
Seoul	Apr. 1-May 31	3	1	
Cuba:	A 120 0 Camb 00	4		First case from S. S. Venezia
Habana	Aug. 2-Sept. 22	•		from Spanish ports arrived
		ł	ł	from Spanish ports; arrived Habana about July 20, 1919 Second case, contact. Fourth
			1	Second case, contact. Fourth
		l	1	case in physician treating pre-
Controller		l	1	vious cases in hospital.
Czecho-Slovakia:	May 18-June 21	11	2	
Prague Denmark:	may 10-3 une 21		_	
Copenhagen	l			Apr. 2-26, 1919: Cases, 11.
Egypt:				• '
Alexandria	May 14-June 24	233	95	
Do	June 25-Aug. 26	199	103 124	
Cairo	Jan. 2-May 20 June 18-July 1	544 158	52	
Do Finland.	Julio 10-July 1	100		Apr. 16-June 30, 1919: Cases, 469.
Provinces—				July 1-15, 1919: Cases, 44.
Abo Och Bjorneborg	Apr. 16-June 30	13		
Kuopio Do	do	88		-
Do	July 1-15	17		
Finland	Apr. 16-June 30	17		
St. Michael	do	73		•
Do Travastchus	July 1-15	63		
Po	July 1-15	5		
V 858	Apr. 16-June 14	12		
Viborg Do	Apr. 16-June 30	340		
Do	July 1-15	36	-	

Reports Received from June 28 to Oct. 17, 1919—Continued.

SMALLPOX—Continued.

Place.	Date.	Cases.	Deaths.	Remarks,
T		+	 	
France: Havre	May 23-30	. 1		<u>.</u>
Marseille	May 1-June 30	:	. 5	
Paris	May 11-June 28	17	i 28	1
Do	June 29-Aug. 30			· .
Gibraltar	June 28-Aug. 16	ï	1 2	One from Bay,
Great Britain:	1	1 -	1	1
Cardiff	June 15-Aug. 30	. 8		.!
Dundee	June 1-7	. 1		
Do	Aug. 18-23	. 9		
GlasgowLiverpool	June 8-21	. 5		
Liverpool	June 22-28	. 1		
Do	June 29-Sept. 6	. 6		
London	May 25-June 28	13		· !
Do	June 29-Aug. 9	18		1
Manchester	July 27-Sept. 6	11		•
Greece:	Mars 15 Turns 00	1	1 40	1
Saloniki	May 15-June 28 June 29-Aug. 23		. 48 43	
DoIndia:	June 29-Aug. 25	1	- 20	į .
Bombay	Apr. 28-June 28	712	283	1
Do	Tuly 6 Aug 2	70	47	1
Calcutta	July 6-Aug. 2 May 4-June 21		444	1
Do	June 29-Aug. 9		109	1 -
Karachi	May 4_Time 91	28	17	ì
Karachi	May 18-June 28 July 6-Aug. 16 Apr. 28-June 28	171	55	Jan. 19-25, 1919: Cases, 29; death:
Do	July 6-Aug. 16	236	107	25.
Rangoon	Apr. 28-June 28	188	92	
Ďo	July 6-Aug. 16	44	20	1
índo China:		i	l	!
Cochin China—		l	1 .	
Saigon	Apr. 21–May 18	11	4	City and district.
taly:			1	
Genoa	July 7-Aug. 31	8		
Leghorn	June 16-29	.2		D
Messina	June 1-21	13	100	Province, June 8-21, 1919: Cases, 23; deaths, 3.
Do Milan	June 29-Aug. 24 Mar. 1-June 30	336	128	23; deaths, 3.
Milazzo.	June 1-7	50 1	î	
Naples	June 2-29	103	91	
Do	June 30-Aug. 17	122	119	
Palermo	May 2-June 20	39	5	
Do	June 28-July 5	37	ğ	
Turin	May 18-June 29	5	i	
Do	June 28–July 5 May 18–June 29 July 6–Sept. 7	8	l	
Venice	May 26-June 1	2	l	
apan:	Ť		1	*
Kobe	May 4-Sept. 7	173	78	
Nagova	June 1-7	1	1	
Taiwan Island	May 21-Aug. 12	20	6	Entire island.
Tokyo	May 1-June 5 May 26-June 1	2		
Yokohama	May 26-June 1	1		
ava:				4 0 7
East Java. Surabaya.		•••••		Apr. 9-June 3, 1919: Cases, 3; July
Mid-Java.	May 27-June 3	2 7		9–15, 1919: Cases, 2.
West Java	Apr. 26-May 16	•		Ma- 0 Toma 00 1010: 0 015
Batavia	Apr 18 Tune 5	4	i	May 2-June 26, 1919: Cases, 615; deaths, 148. June 27-Aug. 25,
Do.	Apr. 18-June 5 July 25-31	3	2	1010: Cases 925: doothe 59
[alta	May 1-31	ĭ	- 1	1919: Cases, 235; deaths, 58.
lanchuria:		-		
Dairen	May 13-June 2	3	2	•
Mukden	July 6-Aug. 23			Present.
[esopotamia:	•			•
Bagdad	May 29-30	1		
lexico:	• ==		1	
Cananea	Feb. 1-28;	7	l	
Do	Apr. 1-30	1		State of Sonora.
Guadalajara	June 1-30	1		· · · · · · · · · · · · · · · · · · ·
Mexico City	June 1-28	20	1	_
Do	June 29-Sept. 6	4		•
l'iedras Negras	June 22-29	2	2	
Salina Cruz	Sept. 1-15	1		
San Jeronimo	June 17-30	. 51		

Reports Received from June 28 to Oct. 17, 1919—Continued.

SMALLPOX-Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
fexico—Continued.				
San Luis Potosi	Sept. 7-13 Sept. 21-27	1	1	
Do	Sept. 21-27	1	2	
Tehuantepec Vera Cruz	Sept. 16	2		
Vera Cruz	July 6-19	4		In State of Oaxaca.
Do	June 29-Aug. 30		9	
Jewfoundland:		-	1	
St. Johns	June 13-Sept. 26	9	1	Tune 13_27 1010 Outnorts 27
	June 15-Sept. 20			June 13-27, 1919: Outports, 37 cases. June 28-Sept. 5, 1919 Cases, 58. Sept. 20-Oct. 3, 1919
Palestine:	Jan. 30-Feb. 5	2		Cases 58 Sant 20 Oct 9 1010
Jaffa	Jami. 30-Feb. 3			Cases, 6.
hilippine Islands:	36 11 17	١,	i i	Casos, v.
Manila	May 11-17	1		•
ortugal:	Tester Of Comt 19	70		
Lisbon	July 26-Sept. 13	78		
Oporto	June 1-28	25	13	
Do	June 29-Aug. 30	66	45	
ortuguese East Africa:		_		
Lourenco Marques	Apr. 1-May 31	2	1	
tussia:	I	ł		
Riga	June 1-30			Present.
iberia:		l		
Vladivostok	June 8-30	45		
Do	July 1-31	12	3	
pain:	i	Ī	i	1
Almeria	May 18-June 30	68	6	
Barcelona	May 15-June 19	3	6	
Do			37	
Bilbao	May 1-10	1		
Cadiz	Apr. 1-May 31 July 1-31	l	5	
Do	Inly 1_31		ž	
Madrid	May 1-31	3	- 1	
Seville	do do		1	
Valencia	May 11-June 29	233	15	
	Tuly 14 Aug 20	73	ii	
Do	July 14-Aug. 30	13		
Vigo	Apr. 12	37	8	
Do	July 6-19	3,		
traits Settlements:	Mar 04 Mars 17	6	1 3	From vessel, Mar. 22, 1919
Singapore	Mar. 24-May 17	5	i	Present in villages in vicinity
	July 8-27	,		Tresent in vinages in vicinity
umatra:	June 26-July 12			Present in surrounding country
Medan	June 20-July 12	• • • • • • • •		Tresent in surrounding country
'unis:	Tuna 15 00	2	1	June 22-28, 1919: Present in sur
Tunis	June 15-28		2	rounding country. June 29
Do	June 29-July 5	3		July 12; Present in surround
nion of South A.rica:	35 1 01			
Johannesburg	May 1-31	1		ing country.
	1			
n vessels:		_	_	D 12 1 G
S. S. Eastern	Apr. 25-26	2	1	Death at sea. Second case land ed at Woodman's Quarantin
	1			ed at woodman's Quarantin
				Station, Fremantie, Australia
	I			Station, Fremantle, Australia Apr. 29. Vessel from Englan
				via Egypt and Colombo. Landed at Colombo. Vess
S. S. Karoa	Apr. 19	1		Landed at Colombo. Vess
	I -			from the United Kingdom vi
•	1			Egypt and Colombo.
S. S. Khyber	Apr. 10-May 4	4	l	From Liverpool, via Port Said
D. D. Mily Dollard		_		Suez, and Colombo. One cas
	!			landed at Port Said Apr. 10
			- 1	Suez, and Colombo. One cas landed at Port Said Apr. 10 2 cases at Colombo Apr. 22, 1 a
	į.			quarantine, Fremantle, Au-
	ŀ			quarantine, Fremantle, Au tralia, May 4, 1919.
C C Die Negree	Oct. 4	1		At Port of Spain, Trinidad, from
S. S. Rio Negros	OC 1. 4	•	• • • • • • • • • • • • • • • • • • • •	Bahia. From Montevideo
				Aug. 31; Santos, Sept. 8; Ri
	1		l	de Janeiro, Sept. 15. Arrive
	1			Port of Spain, Oct. 4, 1919. En route from Naples to Ade
S. S. War Armour		7		En route from Naples to Ade
				and Colombo. Vessel arrive at Fremantle, Australia, Jun
				at Fremantie, Australia, Jun
	l			22, 1919: Cases landed at Co
				lombo.

Reports Received from June 28 to Oct. 17, 1919—Continued.

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Place.	Date.	Cases.	Deaths.	Remarks.
Algeria:				
Algiers	May 1-June 30 July 1-Aug. 31	82	11	
Austria	July I-Aug. 31	1		Mar. 23-Apr. 5, 1919: Cases, 118
Vienna	Mar. 23-Apr. 5	9		
Brazil:	354 7	١.	1	35 00 4 5 1010 5
Rio de Janeiro Do	May 4-June 21 July 6-12	3		Mar. 30-Apr. 5, 1919; Cases, 2.
China:	July 0-12	1 .		
Changsha	May 11-17		1	1
Antung	July 6-30	2		
Chosen (Korea): Chemulpo	Apr. 1-June 30	85	10	
Do	July 1-31	ı	1	
Fusan	May 1-June 30	5	2	1
Do	July 1–31	1	28	·į
SeoulDo	Apř. 1–June 30 July 1–31	147	28	1
Colombia:	July 1-01			
Barranquilla	July 12-19		1	İ
Zecho-Slovakia:	Ma 10 04			
Prague Egypt:	May 18-24	1		
Alexandria	May 14-June 29	474	248	ſ
Do	May 14-June 29 June 28-Aug. 26	431	140	ł
Cairo	Jan. 2-July 1	3, 125	1,796	
Port SaidFinland.	Jan. 9-June 10	11	' '	Apr. 16-June 30, 1919: Cases, 25.
Provinces-			•••••	11pt. 10-sune 50, 1515. Cases, 25.
Abo Och Bjorneborg	May 15	1		
Nyland	Apr. 16-May 31 Apr. 16-June 30	4		
St. MichaelViborg	Apr. 16-June 30	15 3	•••••	
Fermany	Jan. 12-Feb. 22	344		Military.
<u>D</u> o	Feb. 22-Mar. 22	220		Civil.
Do	Mar. 23-Apr. 12	333		Civil, military, prisoners of war,
Do	Apr. 13-26	62		deserters. 55 cases among German troops
. 1	11p 10 20	٠	••••••	and among prisoners of war.
Do	Apr. 27-May 17	126		Of these, 90 among Polish work-
				men and Russians; during same period, 105 cases among Ger-
1		- 1		man troops and prisoners of
1		1		man troops and prisoners of war. In addition, Apr. 1-26, 41 cases were notified among
]	1		41 cases were notified among
reat Britain:	I	- 1		Polish workmen and refugees.
Glasgow	June 8-July 5	13	2	
Dublin	Aug. 17-30	3		June 15-21, 1919: One case.
Dundee				
TOOOO!	June 30-July 5	3		
reece:	1	1	1	
AthensSaloniki	July 21-27. May 15-June 14	3	1 5	
AthensSalonikiDo	July 21-27.	3		71 0 4 0 4 4
reece: Athens	July 21–27. May 15–June 14 July 6–Aug. 23		5 18	Feb. 24-May 9, 1919: Cases, 258.
reece: Athens Saloniki Do ungary Budapest	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5	Feb. 24-May 9, 1919: Cases, 258.
reece: Athens Saloniki Do ungary Budapest	July 21–27. May 15–June 14 July 6–Aug. 23		5 18	Feb. 24-May 9, 1919: Cases, 258. Apr. 28-June 8, 1919: Cases,
reece: Athens Saloniki Do ungary Budapest Debreezin	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners.
reece: Athens Saloniki Do ungary Budapest Debreezin	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,321; Italian soldiers, 82; civi
reece: Athens Saloniki Do ungary Budapest Debreezin	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,321; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Ital-
Athens	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,321; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23: Rumanian soldiers, 23: Rumanian soldiers, 23: Rumanian soldiers, 23: Rumanian soldiers, 24: Rumanian soldiers, 25: Rumanian sol
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23; Rumanian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23; Rumanian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23; Rumanian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23; Rumanian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12 Provinces, with 127 cases, viz, Austrian prisoners, 102; Italian soldiers, 8; civil population, 12; Rumanian soldiers, 8; civil population, 12; Rumanian soldiers, 1, June 23-29, 1919: Present in 14
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,231; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12 Provinces, with 127 cases, viz, Austrian prisoners, 102; Italian soldiers, 8; civil population, 12; Rumanian soldiers, 8; civil population, 12; Rumanian soldiers, 1, June 23-29, 1919: Present in 14
Athens. Saloniki. Do. ungary. Budapest Debreezin. aly. Do.	July 21-27. May 15-June 14. July 6-Aug. 23 Feb. 24-May 9	124	5 18	Apr. 28-June 8, 1919: Cases, 3,470 — Austrian prisoners, 3,321; Italian soldiers, 82; civi population, 67. June 9-15, 1919: Present in 14 Provinces, with 761 cases, viz, Austrian prisoners, 631; Italian soldiers, 23; Rumanian soldiers, 97; civil population, 10. June 16-22, 1919: Present in 12 Provinces, with 127 cases, viz, Austrian prisoners, 102; Italian soldiers, 8; civil population, 12; Rumanian soldiers, 5.

Reports Received from June 28 to Oct. 17, 1919—Continued.

TYPHUS FEVER-Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Italy		1		July 6-13, 1919: Casas 14 annua
16aly		1	1	July 6-13, 1919: Cases, 14, occur- ring in 7 Provinces—7 prisoners
•		ļ		of war, 5 civilians, 2 Italian soldiers.
Do	· ······	· ·····	· ·····	July 21-27, 1919: Cases 5, occurring in 4 Provinces: 1 Aus-
				trian prisoner; 4 civil popula
Do				July 28-Aug. 3, 1919: 6 cases in 3
Genoa Naples	June 25-July 1 May 12-June 22	. 91 50	16	Provinces; civil population.
Palermo	Tuna 20_ A mg 17	1 17	6	1
Venice	Apr. 27-June 14	58	9	
Do Trieste	July 21-27. Apr. 27-June 14. June 30-Sept. 7. June 6-12.	29	5	
Japan: Nagasaki				
Do	July 14-Sept. 7		4	
Mesopotamia: Bagdad	Apr. 19-June 6	34	22	
Do	Apr. 19–June 6 Ly 26–Aug. 1	2		
Guadalajara	May 1-31	1		
Mexico City	May 4-June 28 June 29-Sept. 13	216 272		
San Luis Potosi	July 27-Sept. 27			Present and in surrounding
Newfoundland:				country.
St. Johns Palestine:	June 21-27	1		From vessel.
Jaffa				Oct. 22-Dec. 22, 1918: Cases, 8; deaths, 3.
Portugal:	·			deaths, 3.
Lisbon	June 22–28 July 26–Aug. 23	1 13	2	
Oporto	June 1–15 June 30–Aug. 30	52	42	
Russia:		1 :		
ArchangelRiga	May 15-June 1 May 1-June 30	9 2,826	2	
iberia: Vladivostok	June 9-30	,	9	
Do	July 1-31	56	13	,
Spain: Barcelona	May 15-21		1	
Madrid Sumatra:	May 15–21 May 1–31		1	
Medan	June 26-July 26	2		•
Syria: Mersina	Feb. 13-19			Present.
Mersina Smyrna Cunis:	Feb. 13–19 Sept. 20			Do.
Tunis	May 24-June 21	3	1	
Do	July 20-Aug. 29	. 2	2	
	YELLOW	FEVER	t.	
Brazil:				
Bahia	Apr. 12-June 14	48	15	Jan. 12-May 17, 1919: Cases, 43;
		i		Jan. 12-May 17, 1919: Cases, 43; deaths, 25. July 29, 1919, re- ported seriously prevalent in States of Bahia and Pernam-
	l			States of Bahia and Pernam- buco.
anal Zone	Aug. 10-12	1	1	Patient at Corinto, Nicaragua,
		ļ		at quarantine from S. S. Salva- der.
cuador: Guayaquil	May 1-31	1	1	
Naranjito	May 1-June 15	2	î	July 31, 1919; at Leon, Nicaragua Aug. 2, 1919. Embarked Aug.
Ionduras:				6 at Corinto.
Amapala	Aug. 28	1].		

Reports Received from June 28 to Oct. 17, 1919—Continued.

YELLOW FEVER-Continued.

Place.	Date.	Cases.	Deaths.	Remarks.
Mexico: Merida Nicaragua: Leon.	June 30-Sept. 12 Sept. 1	20	7	State of Yucatan. Present, and in vicinity.
Peru: Department of Piura— Paita	July 10–22	8	5	June 1-Aug. 12, 1919: Cases, 10 deaths, 6.
Piura	July 6 June 24-July 6	46 2		June 1-Aug. 12, 1919: Cases, 90 deaths, 20. 75 miles from city of San Salva-
San Salvador	do	1	1	dor.